The Journal of Bone and Joint Surgery

The Official Publication of
The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association
American Society for Surgery of the Hand
New Zealand Orthopaedic Association
South African Orthopaedic Association

JANUARY 1960

American Volume
Vol. 42-A, No. 1

8 THE FENWAY, BOSTON 15, MASSACHUSETTS, U. S. A.
BRITISH OFFICE:
82 PORTLAND PLACE, LONDON, W. 1, ENGLAND

The Journal of Bone and Joint Surgery

PUBLISHED IN TWO VOLUMES

WILLIAM A. ROGERS, *Editor-in-Chief Emeritus*

AMERICAN VOLUME

Editor and Chairman Board of Editors: THORNTON BROWN

American Board of Associate Editors

CHARLES O. BECHTOL, Los Angeles, California
WILLIAM H. BICKEL, Rochester, Minnesota
DAVID M. BOSWORTH, New York, N. Y.
CRAWFORD J. CAMPBELL, Albany, New York
JONATHAN COHEN, Boston, Massachusetts
ALBERT B. FERGUSON, JR., Pittsburgh, Pennsylvania
S. BENJAMIN FOWLER, Nashville, Tennessee
CHARLES H. FRANTZ, Grand Rapids, Michigan
J. LEONARD GOLDNER, Durham, North Carolina
DAVID S. GRICE, Philadelphia, Pennsylvania
GEORGE HAMMOND, Boston, Massachusetts
CHARLES H. HERNDON, Cleveland, Ohio
FLOYD H. JEROESEN, San Francisco, California
WILLIAM A. LARMON, Chicago, Illinois
LEO S. LUCAS, Portland, Oregon
MICHAEL L. MASON, Chicago, Illinois
JOHN H. MOE, Minneapolis, Minnesota
JOHN ROYAL MOORE, Philadelphia, Pennsylvania
JESSE T. NICHOLSON, Philadelphia, Pennsylvania
FRED C. REYNOLDS, St. Louis, Missouri
DANIEL C. RIORDAN, New Orleans, Louisiana
LEE RAMSAY STRAUB, New York, N. Y
VERNON P. THOMPSON, North Hollywood, California

BRITISH VOLUME

SIR REGINALD WATSON-JONES, *Editor*

British Editorial Board

SIR WALTER MERCER, *Chairman*
H. JACKSON BURROWS, *Deputy Editor*
J. CRAWFORD ADAMS, *Assistant Editor*
W. D. COLTART, *Treasurer*
C. E. L. ALLEN, Cape Town, South Africa
ROLAND BARNES, Glasgow, Scotland
J. S. BATCHELOR, London, England
JOHN CHARNLEY, Manchester, England
F. P. DEWAR, Toronto, Canada
J. KENNEDY ELLIOTT, Wellington, New Zealand
A. L. EYRE-BROOK, Bristol, England
T. J. FAIRBANK, Cambridge, England
ROGER GARIÉPY, Montreal, Canada
B. KRON-COHEN, Melbourne, Australia
DOUGLAS PARKER, Hobart, Tasmania
R. G. PULVERTAFT, Derby, England
ROBERT ROAF, Oswestry, England
H. J. SEDDON, London, England
E. W. SOMERVILLE, Oxford, England
GORONWY THOMAS, Liverpool, England
O. J. VAUGHAN-JACKSON, London, England

Corresponding Editors

Argentina: JOSÉ VALLS
Austria: WALTHER EHALT
Belgium: JEAN VERBRUGGE
Brazil: F. E. GODOY MOREIRA
Cuba: ALBERTO INCLÁN
Denmark: JOHANNES MORTENS
Finland: K. E. KALLIO
France: R. MERLE D'AUBIGNÉ
Germany: MATHIAS HACKENBROCH
Italy: O. SCAGLIETTI
Japan: EISHI KONDO
Mexico: JUAN FARILL
Netherlands: J. D. MULDER
Poland: ADAM GRUCA
Portugal: ARNALDO RODO
Sweden: STEN FRIBERG
Uruguay: JOSÉ LUIS BADO

Committees

THE JOURNAL OF BONE AND JOINT SURGERY, INCORPORATED

The American Orthopaedic Association

The American Academy of Orthopaedic Surgeons

BOARD OF TRUSTEES

DAVID M. BOSWORTH, *Chairman*
KEENE O. HALDEMAN, *Vice-Chairman*
EDWARD L. COMPERE, *Secretary*
H. RELTON MCCARROLL, *Treasurer*
THORNTON BROWN, *Editor*
JAMES S. SPEED
GUY A. CALDWELL

American Society for Surgery of the Hand
President: JULIAN M. BRUNER
President-Elect: DANIEL C. RIORDAN
Secretary-Treasurer: GEORGE S. PHALEN
2020 East 93rd Street, Cleveland 6, Ohio

The Australian Orthopaedic Association
JOHN LAHZ, Brisbane, *President*
WARWICK STENING, Sydney, *Editorial Secretary*
RICHARD HODGKINSON, *Secretary*, 135 Macquarie Street, Sydney, New South Wales

The British Orthopaedic Association
H. J. SEDDON, *President*
SIR REGINALD WATSON-JONES, *Editor*
E. W. SOMERVILLE, *Editorial Secretary*
J. CRAWFORD ADAMS, *Secretary*
47 Lincoln's Inn Fields, London, W.C. 2

The Canadian Orthopaedic Association
W. B. MACKINNON, Winnipeg, *President*
W. W. OLLERHEAD, London, *Vice-President*
IAN W. DAVIDSON, *Secretary-Treasurer*
130 Elm Street, Sudbury, Ontario
F. P. DEWAR, *Editorial Secretary*
706 Medical Arts Building, Toronto, Ontario

New Zealand Orthopaedic Association
H. WALDEN FITZGERALD, Dunedin, *President*
ALAN ALLARD, Dunedin, *Secretary-Treasurer*
N. NISBET, *Editorial Secretary*
Dunedin Hospital, Dunedin, C.1, New Zealand

South African Orthopaedic Association
A. J. HELFET, *President*
SIDNEY SACKS, *Treasurer*
GEORGE DOMMISSE, *Secretary*
303 van Riebeek Medical Buildings, Pretoria, South Africa

Published in the United States by The Journal of Bone and Joint Surgery, Incorporated

Copyrighted in the United States by The Journal of Bone and Joint Surgery, Incorporated

Published in the United Kingdom for The British Editorial Society of Bone & Joint Surgery by E. & S. Livingstone, Teviot Place, Edinburgh

Copyright in Britain and the Commonwealth by The British Editorial Society of Bone & Joint Surgery

THE JOURNAL OF BONE AND JOINT SURGERY

American Office
8 The Fenway, Boston 15, Massachusetts, U.S.A.
Managing Editor: John A. Reed
Assistant Editor: H. Stephen Tilton

British Office
82 Portland Place, London, W. 1, England
Editorial Secretary: Jean Barnes

The Journal of Bone and Joint Surgery*

American Volume

TABLE OF CONTENTS

January 1960

PAGE

Congenital Absence of the Fibula.
By *A. W. Farmer, M.D.*, and *C. A. Laurin, M.D.*, Toronto, Ontario, Canada 1

The Pathomechanics of the Knee Joint. I. The Medial Collateral Ligament and Lateral Tibial Plateau Fractures.
By *Albert F. Martin, M.D.*, Salt Lake City, Utah 13

Acute Suppurative Arthritis of the Hip in the Neonatal Period.
By *Benjamin E. Obletz, M.D.*, Buffalo, New York 23

The Effects of Continuous Compression on Living Articular Cartilage. An Experimental Investigation.
By *Robert B. Salter, M.D., M.S., F.R.C.S.(C).* and *Paul Field, M.B., B.S.*, Toronto, Ontario, Canada 31

Densities of Bones of White and Negro Skeletons.
By *Mildred Trotter, Ph.D., George E. Broman, M.D.*, and *Roy P. Peterson, Ph.D.*, St. Louis, Missouri 50

Clinical and Electromyographic Evaluation of Patients with Anterior Transposition of Peroneal Tendons.
By *William H. Blodgett, M.D.*, and *S. J. Houtz, M.S.*, Detroit, Michigan 59

The Ionization of Metal Implants in Living Tissues.
By *Albert B. Ferguson, Jr., M.D., Patrick G. Laing, M.B., F.R.C.S.*, and *Edwin S. Hodge, Ph.D.*, Pittsburgh, Pennsylvania 77

Heterogenous and Autogenous-Tendon Transplants. An Experimental Study of Preserved Bovine-Tendon Transplants in Dogs and Autogenous-Tendon Transplants in Dogs.
By *J. Edward Flynn, M.D., John T. Wilson, M.D., Charles G. Child, III, M.D.*, and *James H. Graham, M.D.*, Boston, Massachusetts 91

Adductor Myotomy and Obturator Neurectomy for the Correction of Adduction Contracture of the Hip in Cerebral Palsy.
By *Henry H. Banks, M.D.*, and *William T. Green, M.D.*, Boston, Massachusetts 111

(*Continued on page 3*)

The Journal of Bone and Joint Surgery (American Volume) is issued eight times a year—in January, March, April, June, July, September, October, and December. The 1960 subscription price, payable in advance, is $14.50. Single copies $3.00.

The Journal of Bone and Joint Surgery, second-class postage paid at Boston, Mass. and at additional mailing offices. Editorial and business offices, 8 The Fenway, Boston, Mass. Accepted at the special rate of postage provided for in the act of February 28, 1925. Paragraph 4, Section 412, Postal Laws and Regulations; authorized April 21, 1932. Additional entry at Concord, New Hampshire, authorized June 10, 1947.

Copyright 1960 by *The Journal of Bone and Joint Surgery, Incorporated*

*TITLE REGISTERED IN UNITED STATES PATENT OFFICE

The Journal of Bone and Joint Surgery WELCOMES ARTICLES WHICH CONTRIBUTE TO SURGICAL SCIENCE FROM ALL COUNTRIES OF THE WORLD

Manuscripts submitted for publication in the AMERICAN VOLUME should be sent to The Editor, *The Journal of Bone and Joint Surgery*, 8 The Fenway, Boston 15, Massachusetts.
Manuscripts submitted for publication in the BRITISH VOLUME should be sent to The Editor, *The Journal of Bone and Joint Surgery*, 82 Portland Place, London, W. 1, England.

INFORMATION FOR CONTRIBUTORS TO THE AMERICAN VOLUME

- Articles are accepted only for exclusive publication in *The Journal of Bone and Joint Surgery*.
- Publication does not constitute official endorsement of opinions presented in articles.
- Articles published and their illustrations become the property of *The Journal*.

Careful attention to the following suggestions can save much time and correspondence.

WHEN YOU SUBMIT AN ARTICLE PLEASE SEND:

1. THE ORIGINAL MANUSCRIPT. Keep a copy. The original manuscript of an accepted article will not be returned. Several editors report on every manuscript. One or two extra copies with illustrations sent to us will accelerate this procedure.
2. BIBLIOGRAPHY, alphabetical, of references made in text *only*. Please refer to bibliographies in this copy of *The Journal* and follow style exactly.
3. LEGENDS for all illustrations submitted, listed in order.
4. ILLUSTRATIONS
   a. *Black and white glossy prints* of photographs.
   b. *Direct-contact glossy prints* from the original roentgenograms, not prints from secondary negatives.
   c. *Original* drawings or charts.
   d. *Magnification* of photomicrographs.

NOTES ON PREPARATION OF MANUSCRIPT

- Manuscripts must be typewritten, double-spaced with wide margins.
- Write out figures under 100 except percentages, degrees, or figures expressed in decimals.
- Direct quotations should include exact page numbers on which quotation appeared in book or article.

NOTES ON PREPARATION OF ILLUSTRATIONS

- Number all illustrations.
- Indicate *top* plainly.
- Write author's name on the back of each one.
- Put dates or initials in *Legends*, not on prints.
- Send prints unmounted or mounted only with rubber cement. Paste or glue will damage prints.
- Drawings, charts, and lettering on prints should be done with black India ink. Use white India ink on black backgrounds.
- Make lettering large enough to be read when drawings are reduced.
- When submitting an illustration which has appeared elsewhere give full information about previous publication, credit to be given, and state whether or not permission to reproduce has been obtained.

COPYRIGHT

Material appearing in *The Journal* is covered by copyright. As a general rule permission will be given to reputable medical journals to reprint anything in these pages, if permission is first obtained from *The Journal* and if the material used is properly credited to *The Journal*.

FOR ADDITIONAL INFORMATION, PLEASE WRITE TO THE EDITOR

TABLE OF CONTENTS

(Continued from page 1)

PAGE

Pathological Fractures of the Spine. Etiology and Diagnosis. A Review of One Hundred and Five Cases.
By *James A. Nicholas, M.D., Philip D. Wilson, M.D.*, and *Robert Freiberger, M.D.*, New York, N.Y. 127

In Vivo Osteocyte Death.
By *Harold M. Frost, M.D.*, Detroit, Michigan 138

Micropetrosis.
By *Harold M. Frost, M.D.*, Detroit, Michigan 144

Plantar Dissection. An Operation to Release the Soft Tissues in Recurrent or Recalcitrant Talipes Equinovarus.
By *Frederic C. Bost, M.D., Edwin R. Schottstaedt, M.D.*, and *Loren J. Larsen, M.D.*, San Francisco, California 151

Sulphate Metabolism in Cartilage. II. S^{35}-Sulphate Uptake and Total Sulphate in Cartilage Slices.
By *Rose Robyns Coelho, Ph.D.*, and *O. Donald Chrisman, M.D.*, New Haven, Connecticut 165

Late Appearance and Evolution of a Cortical Defect in a Boy with Delayed Puberty.
By *Gerhart S. Schwarz, M.D.*, New York, N.Y. 173

Simple Night Splint for Correction of Tibial Torsion in Children.
By *Rex J. Howard, M.D.*, and *L. P. Kleuser, M.D.*, Fort Worth, Texas 177

Coxa Plana in Monozygotic Male Twins.
By *Commander Adolphus W. Dunn*, Medical Corps, United States Navy 178

Proceedings:
Association of Bone and Joint Surgeons 184

New Jersey Orthopaedic Society 188

Duncan Clark McKeever 189

Book Reviews 191

List of Advertisers ..... Adv. 62

Addresses of Contributors ..... Cover III

Subscription Rates for 1960

American Volume, $14.50 — British Volume, $7.50

Published January, March, April, June, July, September, October, December — Published February, May, August, November

All subscription rates include postage.

Checks should be made payable to *The Journal of Bone and Joint Surgery, Inc.*

Microfilms of the American Volumes are available to regular subscribers through University Microfilms, 313 North First Street, Ann Arbor, Michigan

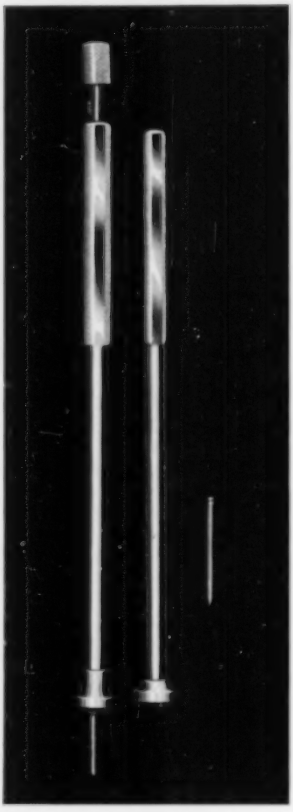
THE JOURNAL OF BONE AND JOINT SURGERY

American Office
8 The Fenway, Boston 15, Massachusetts, U. S. A.

British Office
82 Portland Place, London, W. 1, England

NEW... FROM Orthopedic

SMILLIE'S
nail gun
AND
nail punch



Now, these superb appliances developed for treatment of osteochondritis dissecans of the knee are available thru your surgical supply dealer.

No. 437—Smillie's Nail Gun—Stainless steel throughout. Light in weight and carefully balanced.

No. 436—Smillie's Nail Punch—Stainless steel.

No. 438—Smillie's Nails—are SMo stainless steel, 5/8", 3/4", 7/8", 1" and 1 1/4" long. Diameter 1/16". All have grooved head for ease of extraction.

Set has other uses including internal fixation of recent fractures of the articular surfaces of joints, fractures in the hand, Bennett's fracture, fractures of the phalanges, carpo-metacarpal arthrodesis, etc.

Facilitates the insertion of Smillie's SMo Stainless Steel Nails in the treatment of Osteochondritis Dissecans.

A hammer tap on the plunger, which protrudes from the gun, insures effective insertion.

Complete insertion of the nail is achieved by impacting with punch, which has a small recess in head to take nail.

Order now, from your surgical supply dealer!

Plants also in Great Britain and Germany

Orthopedic EQUIPMENT CO.
BOURBON · INDIANA

SPLINTS · FRACTURE EQUIPMENT · BONE INSTRUMENTS
SMO INTERNAL BONE APPLIANCES · ORTHOPEDIC SUPPLIES

remember this patient with musculoskeletal pain?

PAIN remember Zactirin®

Ethoheptazine Citrate (75 mg.) with Acetylsalicylic Acid (325 mg.), Wyeth

Active now, pain relieved, inflammation controlled, *free of disturbing reactions*. . . . ZACTIRIN offers reliable analgesia plus anti-inflammatory benefits in a wide variety of joint and muscle disorders. It is non-narcotic and nonsteroid. Its relief of pain is equivalent to that of codeine. Yet it is well tolerated in both acute and prolonged use.

Myositis, fibrositis, myalgia, low back pain, ligamental strain, sciatica, bursitis, frozen shoulder, wryneck, osteoarthritis, rheumatoid arthritis, postoperative orthopedics

Supplied: Tablets, bottles of 48.

Wyeth®
Philadelphia 1, Pa.

SAFER and QUICKER MENISECTOMIES

with BOWEN Instruments

Grover Meniscotome

Used through any arthrotomy exposure and on either semi-lunar cartilage, it guarantees the integrity of all structures within the joint and permits complete removal of either meniscus.

Designed like a large intra-uterine curette, it presents a smooth rounded guard on the outer surface of the ring, the inner margin of which is a keen knife edge permitting cutting in any direction. Its use will shorten by about one-third the present operating time.

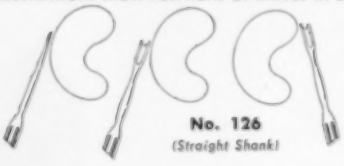
Standard Length: 8½" • **No. 128**

Smillie Cartilage Sets

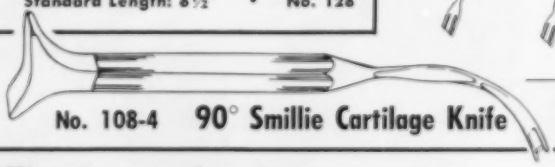
Designed with handles that do not obscure field of vision. Longer beak always rests on tibial table. Beaked knife divides the peripheral attachments of the medial meniscus. Also divides the central attachment of the posterior horn of the lateral meniscus. Second beaked knife used for the same purpose, also to divide the central attachment of the posterior horn of the medial meniscus. Chisel mobilizes middle third of the meniscus.

No. 108
(Curved Shank)

ILLUSTRATIONS SHOW POSITIONS OF KNIVES IN USE



No. 126
(Straight Shank)



No. 108-4 90° Smillie Cartilage Knife

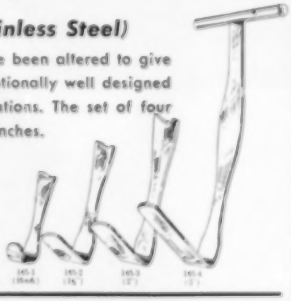
A specially curved shank to facilitate the removal of the posterior horn of the meniscus.

Smillie Knee Joint Retractor Set *(Stainless Steel)*

The angles of the blades and the modified handles of these retractors have been altered to give a better grip and keep the fingers away from the incision. They are exceptionally well designed for the removal of menisci and for use in practically all knee joint operations. The set of four comprises one hook and three with blade lengths from 1½ inches to 3 inches.

FEATURES: "Sure grip handles for perfect control." • "Hardened and tempered to rigid specifications." • "Useful retractors for many other surgical procedures."

No. 145 Knee Joint Retractor Set
(Four Pieces)



New!

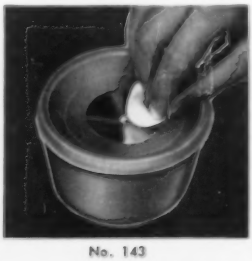
Unique!

BOWEN Liquid DISPENSER

For the dispensing of alcohol and other cleansing liquids.
Patent No. 2,752,069

- *Quick Acting • Unbreakable • Will Not Tip*
- *Easily Operated with one hand*
- *To Use, Just Press on Metal Plate!*

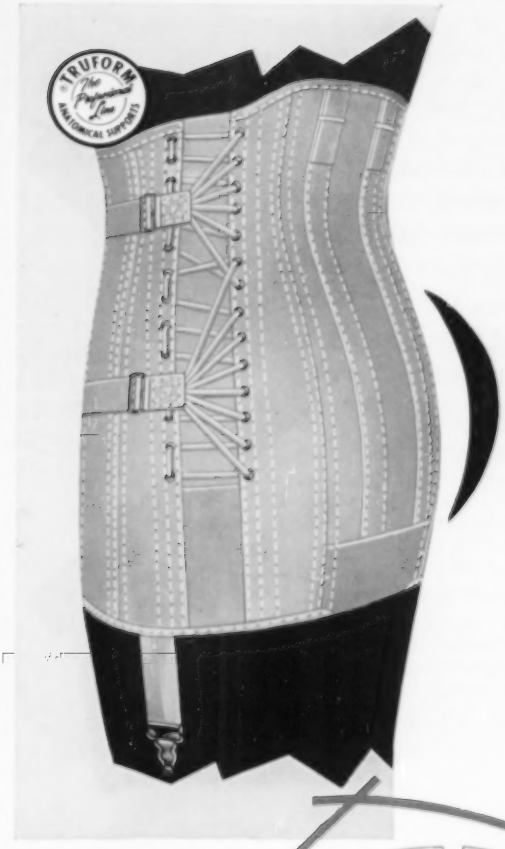
ONLY $1.50



No. 143

BOWEN & COMPANY, INC.

P. O. BOX 5818
BETHESDA, MARYLAND



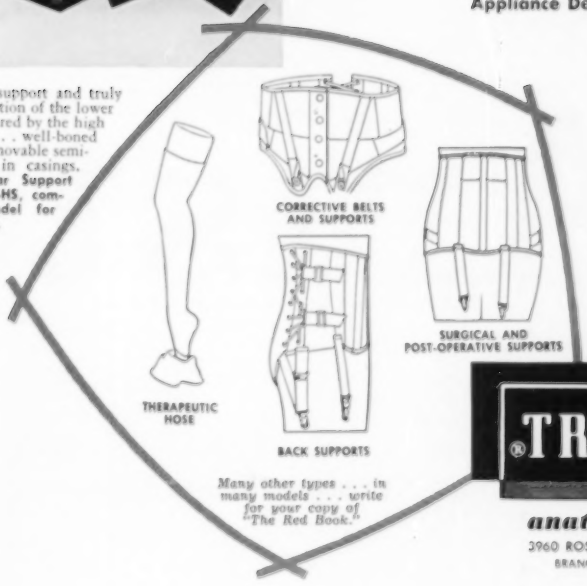
effective FIXATION

Anatomically correct . . . therapeutically sound . . . these principles are designed in, and built in, each Truform appliance.

Effective support or effective fixation . . . this you can depend on when you prescribe "Truform." Your patient benefits from the most careful selection and skillful fitting because . . .

- **TRUFORM Anatomical Supports are available only from the Ethical Appliance Dealer**

Very rigid support and truly effective fixation of the lower spine is assured by the high solid back . . . well-boned and with removable semi-rigid stays in casings. **Sacro-Lumbar Support Model 1165-HS, comparable model for men 447-HS.**



Many other types . . . in many models . . . write for your copy of "The Red Book."

TRUFORM

anatomical supports

3960 ROSSLYN DRIVE, CINCINNATI 9, OHIO

BRANCHES: New York and San Francisco

when the
rheumatic
disorder
is more
than salicylates
alone
can control...

MORE
HIGHLY INDIVIDUALIZED
THERAPY
FOR THE
RHEUMATIC
"IN-BETWEEN"

...but
control
requires less
than
intensive
steroid therapy
alone

Aristo

wider latitude in adjusting dosage

ARISTOGESIC is particularly effective for relief of chronic – but less severe – pain of rheumatic origin. ARISTOGESIC combines the anti-inflammatory effects of ARISTOCORT® Triamcinolone with the analgesic action of salicylamide, a highly potent salicylate. Dosage requirements for ARISTOGESIC are substantially lower than generally required for each agent alone. The exceptionally wide latitude of dosage adjustment with ARISTOGESIC permits well-tolerated therapy for long periods of time with fewer side effects.

Indications: Mild cases of rheumatoid arthritis, tenosynovitis, synovitis, bursitis, mild spondylitis, myositis, fibrositis, neuritis, and certain muscular strains.

Dosage: Average initial dosage: 2 capsules 3 or 4 times daily. Maintenance dosage to be adjusted according to response.

Precautions: All precautions and contraindications traditional to corticosteroid therapy should be observed. The amount of drug used should be carefully adjusted to the lowest dosage which will suppress symptoms. Discontinuance of therapy must be carried out gradually after patients have been on steroids for prolonged periods.

Each ARISTOGESIC Capsule contains:

ARISTOCORT® Triamcinolone	0.5 mg.
Salicylamide	325 mg.
Dried Aluminum Hydroxide Gel	75 mg.
Ascorbic Acid	20 mg.

Supply: Bottles of 100 and 1,000.

gesic® Capsules

Steroid-Analgesic Compound LEDERLE

Lederle

LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

PROGRESSIVE LEADERSHIP
...IN BASIC FOOTWEAR

EQUINO-VARUS

Wide 000 to 4
Narrow 0000 to 4

The foot is enveloped in a smooth seamless upper. The shoe keeps the foot in the exact position the physician has obtained. Open toe permits free circulation. Steel plate between inner and outersole permits application of corrective force at point desired.

SURGICAL

Meets the need of a matching shoe where only one foot requires the Equino-Varus shoe. Made on straight last.

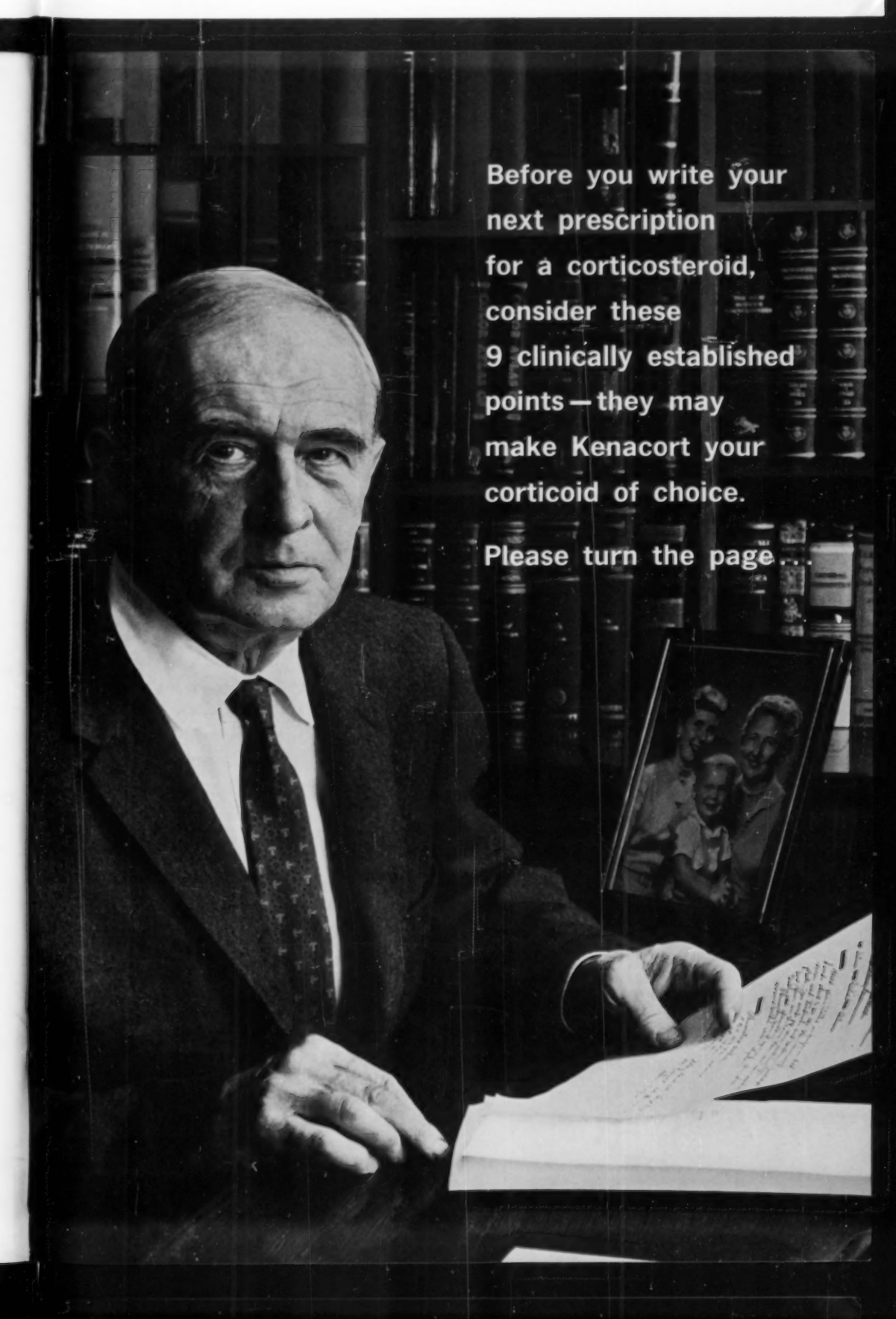
Medium 000 to 4

Roto-Lok disc inserted in bottom of shoe... allows more accurate setting, is located in proper spot, reduces overall cost of shoe and brace. Interchangeable with Fillauer Night Split.

Both carried in stock.

SABEL'S
EQUINO-VARUS
TRADE MARK REG. U.S. PAT. OFF.

by R. J. Potvin Shoe Co., Brockton, Mass.


Before you write your
next prescription
for a corticosteroid,
consider these
9 clinically established
points — they may
make Kenacort your
corticoid of choice.
Please turn the page . . .

for choice corticosteroid therapy

consider...

pre-prescription point number **1**

initial therapy—remarkably free from complications

Allison, J. R., Sr., and Allison, J. R., Jr.: Monographs on Therapy 3:99 (Oct.) 1958.

pre-prescription point number **2**

continuing therapy—maintenance doses are low

Feinberg, S. M.; Feinberg, A. R., and Fisherman, E. W.: J. A. M. A. 167:58 (May 3) 1958.

pre-prescription point number **3**

no sodium or water retention—low salt diet not necessary

Hartung, E. F.: J. A. M. A. 167:973 (June 21) 1958.

pre-prescription point number **4**

absence of edema

Council on Drugs: J. A. M. A. 169:257 (Jan. 17) 1959.

pre-prescription point number **5**

less likely to create electrolyte disturbance

Bongiovanni, A. M.; Mellman, W. J., and Eberlein, W. R.: J. Pediat. 53:3 (July) 1958.

pre-prescription point number **6**

no secondary hypertension—no significant change in pulse, respiration, or blood pressure

Shelley, W. B.; Harun, J. S., and Pillsbury, D. M.: J. A. M. A. 167:959 (June 21) 1958.
Bernstein, C. A., Jr., and others: New York Rheumatism Association, Annual Meeting, New York, April 9, 1959.

Kenacort

(SQUIBB TRIAMCINOLONE)

Available in 1 mg., 2 mg., and 4 mg. scored tablets.

pre-prescription
point number **7**

no excessive appetite

Council on Drugs:
J. A. M. A. 169:257 (Jan. 17) 1959.

pre-prescription
point number **8**

without unnatural psychic stimulation —does not stimulate and rarely depresses the mood

Shelley, W. B.; Harun, J. S., and Pillsbury, D. M.:
J. A. M. A. 167:959 (June 21) 1958.
Council on Drugs:
J. A. M. A. 169:257 (Jan. 17) 1959.

pre-prescription
point number **9**

gastrointestinal complaints infrequent

Hartung, E. F.:
J. A. M. A. 167:973 (June 21) 1958.

SQUIBB

Squibb Quality — the Priceless Ingredient

'KENACORT'® IS A SQUIBB TRADEMARK

...THEY CAN WALK AGAIN

Torpedoed on the Murmansk run—nearly frozen to death in an open boat—both legs lost below the knee—ex-Merchant Marines Michael McCormick and William Morris walked unaided in three weeks. They could look forward with certainty to leading a normal life again. To these men, as to thousands of other Hanger wearers, the phrase "Hanger is a symbol of help and hope" is a concrete truth proven by every day of their future lives.

Hanger PROSTHESIS

AVAILABLE AT AUTHORIZED FACILITIES IN THE FOLLOWING CITIES

Eastern Region:
Baltimore 1, Md.
Boston 15, Mass.
Charleston 2, W. Va.
New York 11, N. Y.
Philadelphia 7, Pa.
Raleigh, N. C.
Richmond 19, Va.
Roanoke 12, Va.
Washington 13, D. C.

Midwestern Region:
Chicago 5, Ill.
Cincinnati 29, Ohio
Dallas 1, Texas
Evansville, Ind.
Fort Wayne, Ind.
Houston, Texas
Indianapolis 2, Ind.
Oklahoma City 3, Okla.
Peoria 4, Ill.
St. Louis 66, Mo.
Wichita, Kansas

Central Region:
Columbus 8, Ohio
Johnstown, Pa.
Pittsburgh 1, Pa.

Southeastern Region:
Atlanta 9, Ga.
Birmingham 1, Ala.
Columbia 5, S. C.
Jacksonville, Fla.
Miami 37, Fla.
Mobile, Ala.
Montgomery, Ala.
Nashville 5, Tenn.
New Orleans 19, La.
Orlando, Fla.
Savannah, Ga.
St. Petersburg, Fla.
Tampa 2, Fla.
West Palm Beach, Fla.

In answering advertisements, please mention *The Journal of Bone and Joint Surgery.*

NEW Fillauer

DETACHABLE

DENIS BROWNE NIGHT SPLINT

STREAMLINED and IMPROVED

1. Precision die cast Serrated Discs permit 50 positions.
2. New Knurled Screw locks discs in position.
3. New Triple Flange fits club foot shoes or any sole thickness.
4. Streamlined Wing Screw secures flange to shoe sole.
5. Durable, attractive red alumilite color on bars.
6. Lengths 6, 7, 8, 9, 10, 12, 14, 16 and 18 inches. Special lengths to 30 inches at no extra cost.

ORTHOPEDIC SURGEONS recognize the decided advancements this versatile splint affords in treating club feet, positional deformities, tibial torsions, flat feet and congenital hip dislocations. Also applicable for any other deformities that are actively corrected by incessant kicking.

Very little adjustment by surgeon or orthotist is required to fit and position this new Fillauer Detachable Splint.

Pre-walker or larger surgical and club foot shoes work extremely well with the Fillauer Splint. Outgrown shoes may be used by cutting out toes.

Fillauer SURGICAL SUPPLIES, INC.
BOX 1678 CHATTANOOGA, TENN.
ESTABLISHED 1914 *Manufacturers of Orthopedic Supplies*

relieves painful muscle spasm, improves mobility, facilitates rehabilitation...

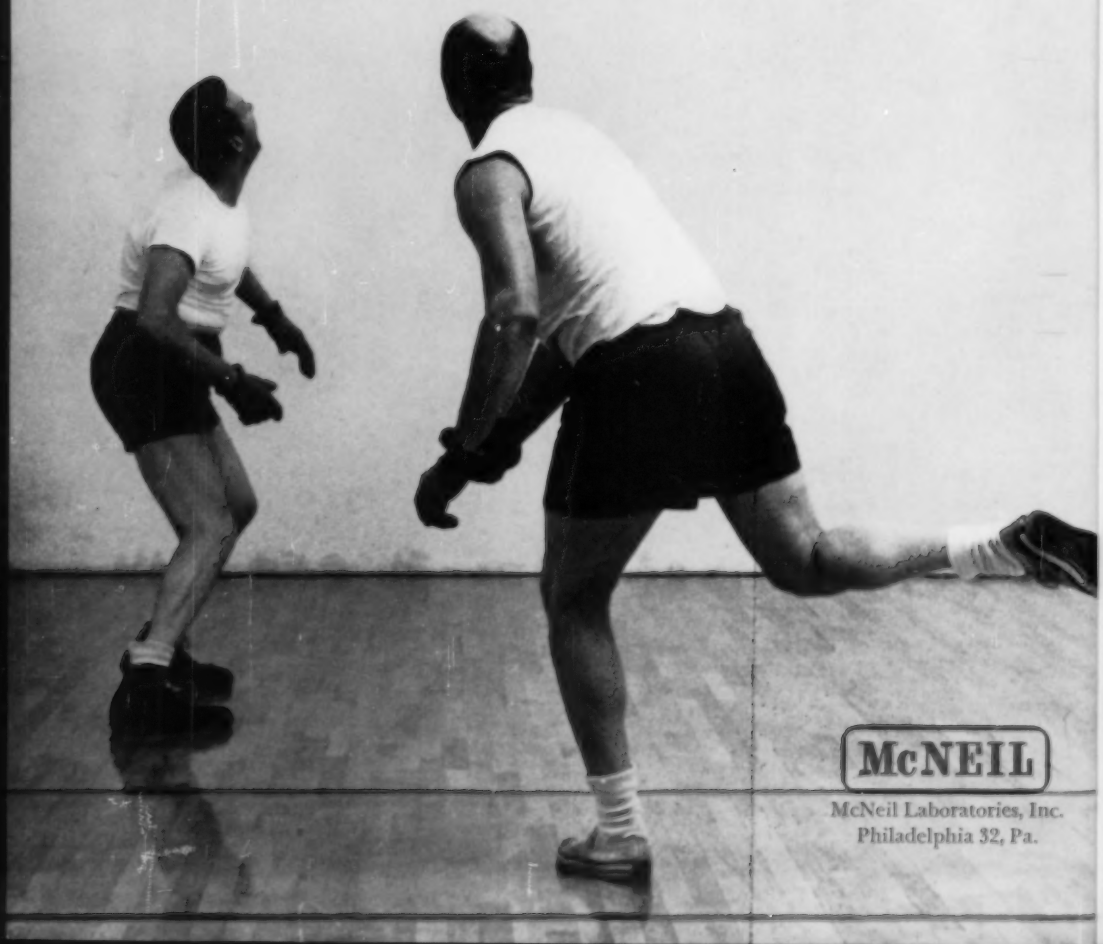
PARAFLEX®

Chlorzoxazone*

PARAFLEX provides effective skeletal muscle relaxation for about 6 hours with a 1- to 2-tablet dose. It relieves pain and stiffness and improves function in a wide variety of orthopedic, arthritic, and rheumatic disorders. It may be used alone or with other agents indicated in the management of skeletal muscle spasm. It is especially valuable when used in conjunction with physiotherapy and other rehabilitative procedures. Side effects are rare, almost never require discontinuance of therapy.

Dosage: ADULTS—1 to 2 tablets three or four times a day.
CHILDREN—½ to 2 tablets three or four times a day, depending on age and weight.
Supplied: Tablets, scored, orange, bottles of 50. Each tablet contains PARAFLEX, 250 mg.

*U.S. Patent Pending 255A59

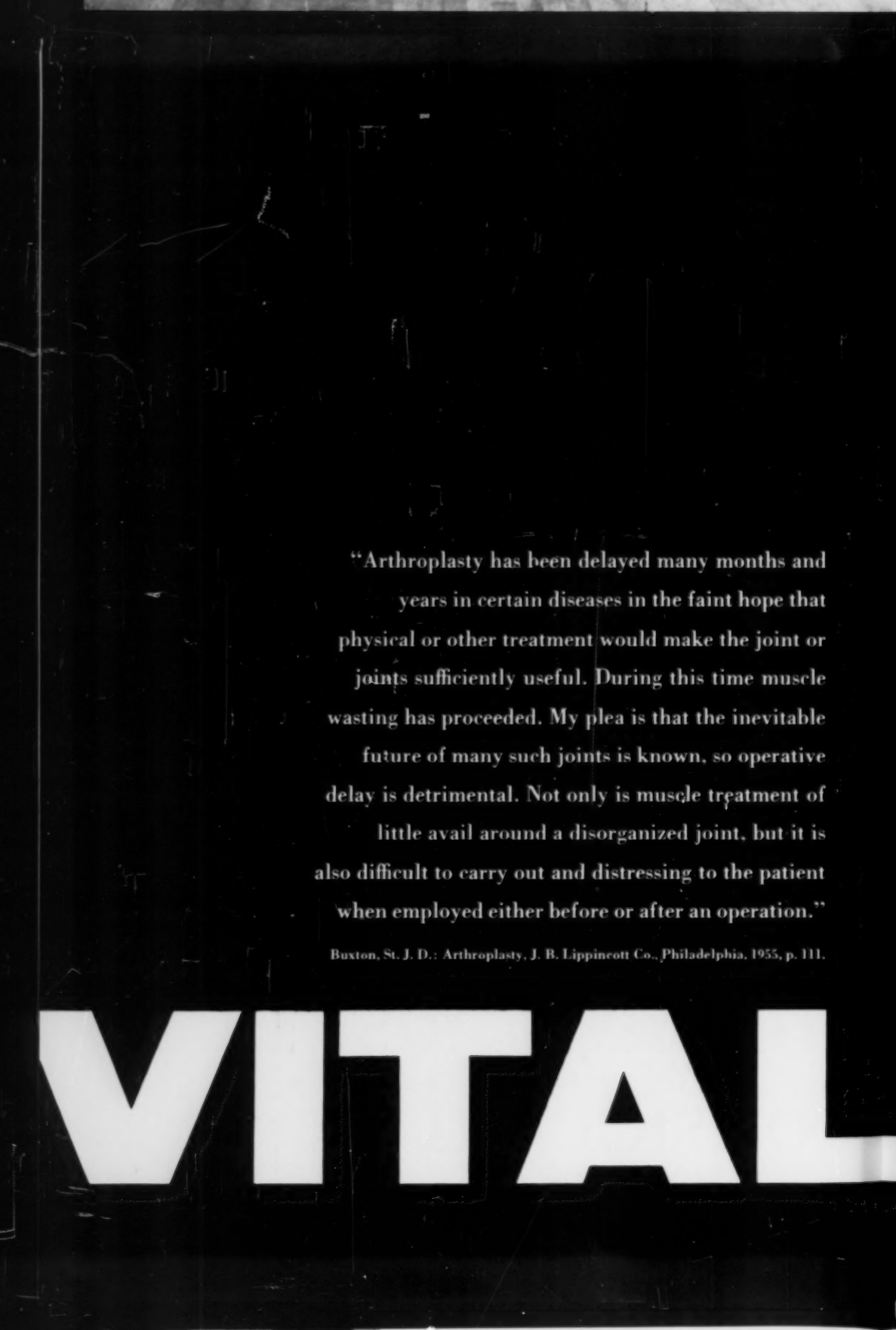


McNEIL

McNeil Laboratories, Inc.
Philadelphia 32, Pa.

"when conservative measures for alleviation of pain and/or deformity of the osteoarthritic hip become ineffectual, surgery must be considered"

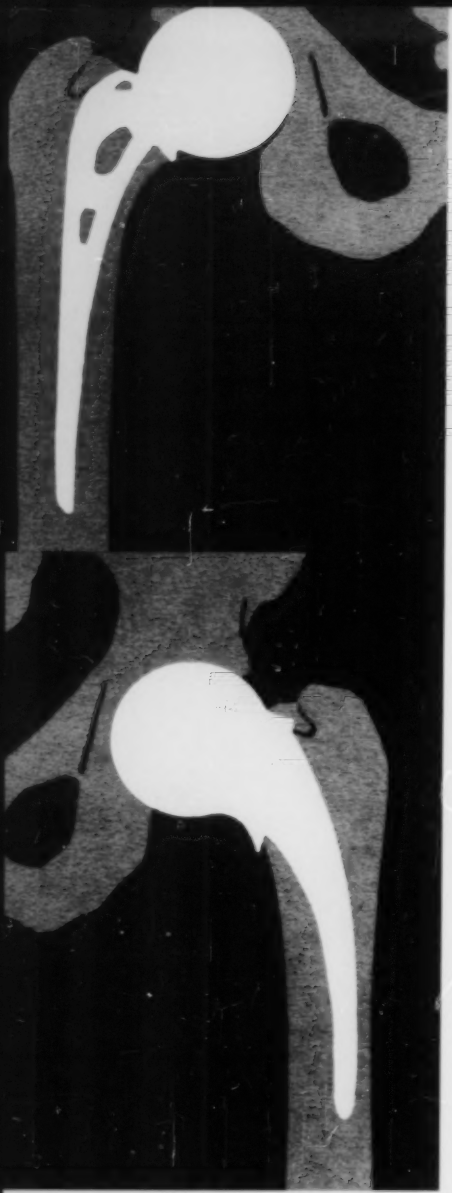
Vare, V. B.: Medical Forum, Mod. Med. 27:33, 1959.


"Arthroplasty has been delayed many months and years in certain diseases in the faint hope that physical or other treatment would make the joint or joints sufficiently useful. During this time muscle wasting has proceeded. My plea is that the inevitable future of many such joints is known, so operative delay is detrimental. Not only is muscle treatment of little avail around a disorganized joint, but it is also difficult to carry out and distressing to the patient when employed either before or after an operation."
Buxton, St. J. D.: Arthroplasty, J. B. Lippincott Co., Philadelphia, 1955, p. 111.
VITAL

"The replacement prosthesis has a very definite place in surgery of the arthritic hip, especially when the head of the femur is essentially gone or inadequate."

Aufranc, O. E.: Medical Forum, Mod. Med. 27:33 1959.

LIUM



"Vitallium appliances are manufactured with exacting care and precision. Each one is x-rayed and carefully tested for flaws. Composition of the metal is standardized. It has been proved to be inert and nonelectrolytic in the tissues. Vitallium . . . lends itself more easily to the manufacture of the Moore prosthesis than does stainless steel which requires the process of machining. Vitallium is the author's material of choice and theoretically, it may last indefinitely and give satisfactory service throughout a patient's expected life span."

Moore, A. T.: *In* Am. Acad. Orthop. Surg., Instructional Course Lectures, v. 16, C. V. Mosby, 1959, p. 309.

"Vitallium . . . has proved superior to the use of steel or other metals or plastics. Not only is Vitallium a satisfactory metal, but the company producing the appliances is using more than ordinary skill, care, and know-how, to produce a dependable product."

Thompson, E. R.: *In* Am. Acad. Orthop. Surg., Instructional Course Lectures, v. 16, C. V. Mosby, 1959, p. 299.

where femoral
head replacements
are indicated
specify

VITALLIUM®

HIP PROSTHESES

AUSTENAL COMPANY
DIVISION OF HOWE SOUND COMPANY

VITALLIUM® *Surgical* PRODUCTS
224 EAST 39TH STREET
NEW YORK 16, NEW YORK

Your patients with LOW BACK PAIN can expect striking relief and return to normal activity when you prescribe *Trancopal*

case profile.[1] A 42-year-old truck driver and mover injured his back while moving a piano. The pain radiated from the sacral region down to the region of the Achilles tendon on the right side. X-rays for ruptured disc revealed nothing pertinent. The day of the injury he was given Trancopal immediately after the physical examination. Although 100 to 200 mg. three times a day were prescribed, the patient on his own responsibility increased the dosage of Trancopal to 400 mg. three times a day. This dosage was continued for three days and then gradually reduced over a ten day period. During this time, the patient continued to drive his truck. The muscle spasm was completely controlled and no apparent side effects were noted. For the past six months, the patient has continued to take Trancopal 100 to 200 mg. as needed for muscle spasm, particularly during strenuous days.

Indications — Musculoskeletal: Neck pain (torticollis) / Ankle sprain, tennis elbow / Bursitis / Rheumatoid arthritis / Low back pain (lumbago, etc.) / Fibrositis / Myositis / Osteoarthritis / Postoperative muscle spasm / Disc syndrome. *Psychogenic:* Dysmenorrhea / Anxiety and tension states / Asthma / Premenstrual tension / Angina pectoris / Alcoholism.

THE FIRST TRUE "TRANQUILAXANT"

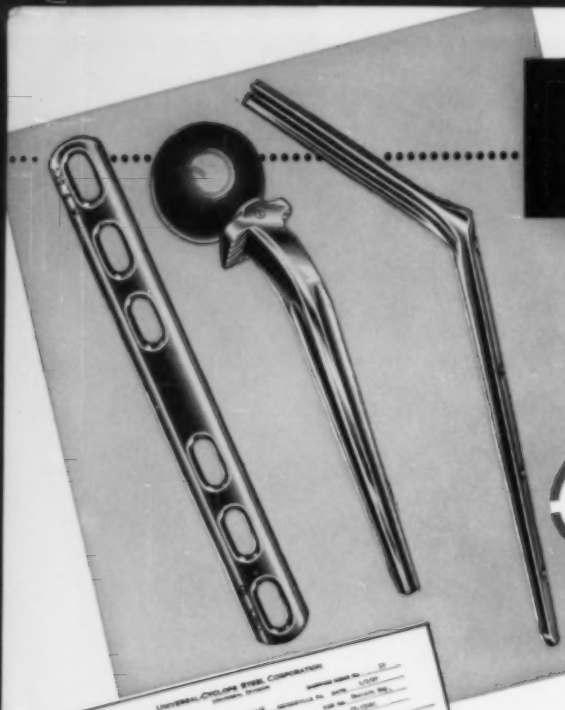
Trancopal®

Winthrop LABORATORIES • NEW YORK 18, N.Y.

Now available in two strengths: Trancopal Caplets®, 100 mg. (peach colored, scored), bottles of 100. **New strength**—Trancopal Caplets, 200 mg. (green colored, scored), bottles of 100.
Dosage: Adults, 100 or 200 mg. orally three or four times daily. Relief of symptoms occurs in from fifteen to thirty minutes and lasts from four to six hours.

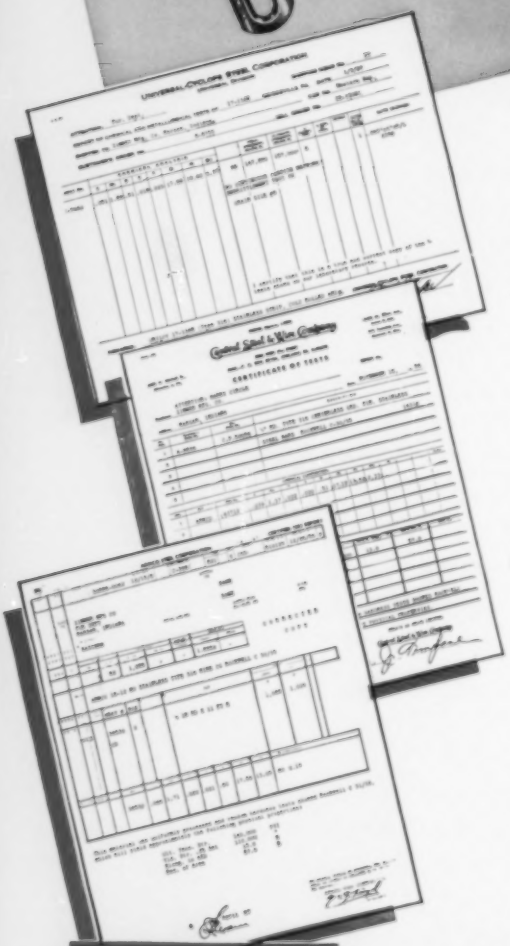
1. Collective Study, Department of Medical Research, Winthrop Laboratories.

Trancopal (brand of chlormezanone) and Caplets, trademarks reg. U.S. Pat. Off. 14164M



zimmer®

stainless steel CERTIFIED for uniformity



Every bar of stainless steel that goes into the manufacture of Zimmer orthopedic appliances and equipment is certified for uniformity of physical properties by the metallurgical laboratories of some of America's foremost steel mills.

Armco, Universal Cyclops and Central Steel & Wire Company all cooperate closely by furnishing certifications with each shipment. Elements alloyed in Zimmer stainless steels vary less than 1% from one production heat to another.

Such uniformity assures the orthopedic surgeon of utterly dependable implant appliances – appliances tested to resist the destructive actions of body fluids. Zimmer metals are unsurpassed.

zimmer®

ZIMMER MANUFACTURING CO. • WARSAW, INDIANA, U.S.A.

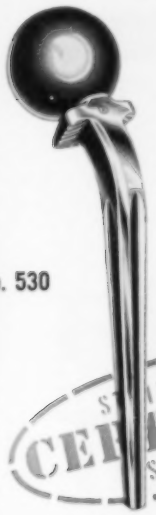
LOOK FOR THE TRADEMARK (Z)

appliances for orthopedic surgery

zimmer®

THE EICHER FEMORAL PROSTHETIC HEAD

No. 530



TOWNLEY PROSTHESIS

No. 815

MINNEAPOLIS PROSTHESIS

No. 535

SCHNEIDER SELF-BROACHING INTRAMEDULLARY PIN

No. 825

SLOTTED PATTERN PLATES

No. 539

ADJUSTABLE INTERTROCHANTERIC PLATE

McLAUGHLIN TYPE

ELLIOT FEMORAL CONDYLE BLADE PLATE

No. 554

No. 560
(Nail Not Included)

ZIMMER MANUFACTURING CO. • WARSAW, INDIANA, U.S.A.

zimmer®
LOOK FOR THE TRADEMARK

CAMP TRADE MARK *sacro-iliac supports*

For Conservative Treatment of Low Back Syndromes

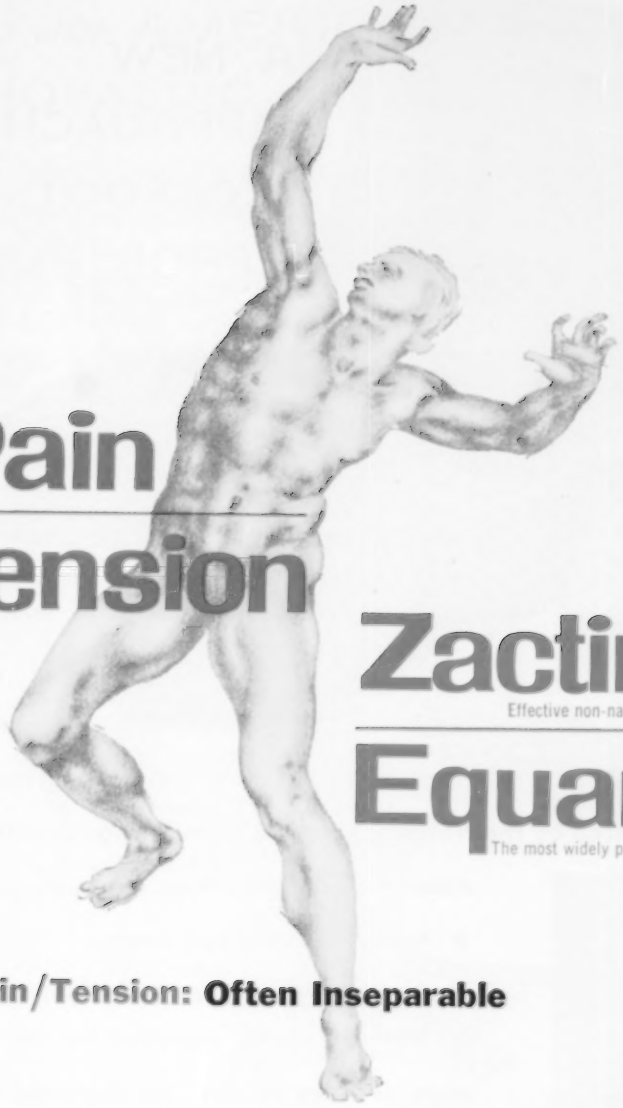
This front-closing, back-lacing men's support has a pad laced into the back to apply pressure where it is most needed. The buckle-lacing adjustment feature brings the garment firmly around the pelvis to rest and support the sacral joints.

Women with sacro-iliac trouble have found this style garment offers them relief and help from backache. Camp's "block-and tackle" double adjustment back-lacing feature snugs the garment firmly around the pelvis. Elastic inserts top and bottom at sides and lower center front expand to assure the wearer garment comfort.

Many orthopedic surgeons have come to recognize Camp sacro-iliac supports as an invaluable aid in the treatment of strain and sprain in sacro-iliac conditions. The basic design concept in all Camp sacro-iliac supports is to firmly encircle the bony pelvis between the crests of the ilia and the trochanters, while also providing support to the erector spinae muscles. Often these supports are recommended for daily wear as a preventive measure for patients chronically bothered by backache. These supports are all made of firm fabrics and are well boned. Pads can be inserted where prescribed.

S. H. CAMP & COMPANY, JACKSON, MICHIGAN CAMP

S. H. Camp & Company of Canada, Ltd., Trenton, Ontario



Pain
Tension

Zactirin®*
Effective non-narcotic analgesia

Equanil®†
The most widely prescribed ataraxic

Pain/Tension: Often Inseparable

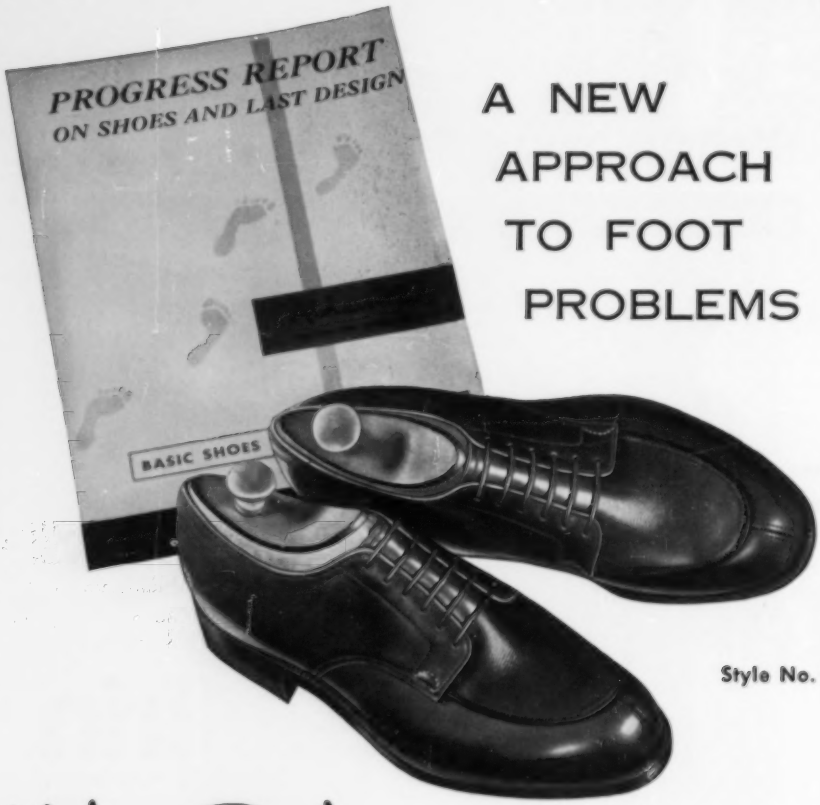
ZACTIRIN and EQUANIL administered together produce relaxation from tension—muscular and mental—and relief from pain.

EQUANIL enhances the action of ZACTIRIN by reducing the tension which often accompanies pain and increases awareness of it. *Wyeth Laboratories, Philadelphia 1, Pa.*

*Ethoheptazine Citrate with Acetylsalicylic Acid, Wyeth †Meprobamate, Wyeth

100 ANNIVERSARY Wyeth

A Century of Service to Medicine



A NEW APPROACH TO FOOT PROBLEMS

Style No. 84

Alden-Pedic SCIENTIFIC CONSTRUCTION SHOES

Preferred by the Nation's Leading Foot-Fitting Specialists

- Long inside counter
- Right and left ortho heels, long inside
- Heavy gauge right and left ribbed steel shank.
- New "Depth Design" accommodates prescribed corrections

WRITE FOR BOOKLET AND NAME OF NEAREST DEALER

Alden-Pedic lasts and shoes serve 3 purposes:

- Fit the individual foot shape . . .
- Accommodate your prescribed corrections . . .
- Provide a stable, controlled foundation.

Write us today for our new illustrated brochure of Alden-Pedic styles for men and boys and our new "Progress Report on Shoe and Last Design."

C.H. ALDEN SHOE COMPANY

Custom Bootmakers Since 1884

BROCKTON, MASSACHUSETTS

Precise Positioning and Positive Fixation—

with AMERICAN-Albee

ORTHOPAEDIC *and* FRACTURE TABLE

● Acknowledged as the most versatile in its field, the American-Albee Orthopaedic and Fracture Table enables the orthopaedic surgeon to achieve "perfect positioning" of short, tall, obese and even infant patients . . . easily and rapidly. It is designed to accommodate closed fracture reductions as well as the increasing demands of complex orthopaedic surgery. Thus, to orthopaedists the world over, the name American-Albee identifies the table of choice for this comprehensive branch of medicine.

Ideal complement to this improved table is the American DV-22 Major Surgical Light . . . a new concept in easily maneuverable illumination . . . cool, shadowless, color-corrected . . . intensity to 10,000 foot candles *from a dual source.*

● *Write for Fracture Table Brochure TC-100R and Major Light Catalog LC-121R.*

Offices in 14 Principal Cities

AMERICAN STERILIZER

ERIE • PENNSYLVANIA

ANNOUNCING SCHERING'S NEW MYOGESIC[X]

RELA™ **EASES STRAINS SPRAINS & LOW BACK PAINS...!**

X

CARISOPRODOL

RELA—a new myogesic for better relaxant *and* analgesic therapy—more adept management of spasm and pain in strains, sprains and low back pains.

RELA—though a single drug—is a true myogesic and works rapidly to achieve three desired effects...

Rela relaxes acute muscle spasm
Relief of muscle spasm (96% excellent to good effectiveness)[1]

Rela provides a unique quality of persistent pain relief through its relaxant and analgesic actions
"Relief from pain was usually rapid and sometimes dramatic"[1]

Rela, through relaxation and analgesia, assures daytime ease and nighttime rest
"... A number of patients reported freedom from insomnia which they attributed to freedom from pain."[1]

indications: RELA is most beneficial in those conditions of the musculoskeletal system manifesting pain, stiffness and spasm.

safety: Studies of more than 1400 patients indicate that the toxicity of RELA is exceptionally low. In human subjects, respiratory, blood pressure or blood chemistry changes and/or renal, hepatic or endocrine dysfunction have not been reported.

dosage: The usual adult dosage of RELA is one tablet 3 times daily and at bedtime. RELA has a rapid onset of action, with relief usually apparent within 30 minutes, and persisting for at least 6 hours.

supply: RELA is available as 350 mg., pink, coated tablets in bottles of 30.

1. Ruge, T.: To be published.

MYOGESIC
muscle-analgesic relaxant

Schering

the action of the TARSO SUPINATOR SHOE

. . . swings the hind-foot inward, which induces adduction of the fore-foot, elevates the longitudinal arch, and levels the ankle joint.

When the hind-foot is held in a position of inward rotation, the talo-scaphoid and cuneiform-scaphoid joints will remain locked under weight bearing. The arch will not collapse and the ankle will not roll inward.

Correction is accomplished by the specially shaped last. Medially wedged and extended heels promote better weight distribution. Medially extended counters are used for utmost control.

See us in Chicago — January 23–28
27th Annual Meeting
American Academy of Orthopaedic Surgeons
BOOTH 30

BEFORE **AFTER**

The Tarso Supinator shoe is used successfully in treatment of the plano-valgus foot. Pronation is controlled at once. Permanent improvements in walking and in foot posture will generally occur. Patients find Tarso Supinators notably comfortable.

Tarso Shoes are ready-made and are available on prescription from dealers in most cities. Write for catalog and name of nearest dealer.

TARSO SUPINATOR REG U.S. PAT. OFF. SHOE
FOR FLAT FEET

M. J. MARKELL SHOE COMPANY, INC.

332 SOUTH BROADWAY • YONKERS, NEW YORK

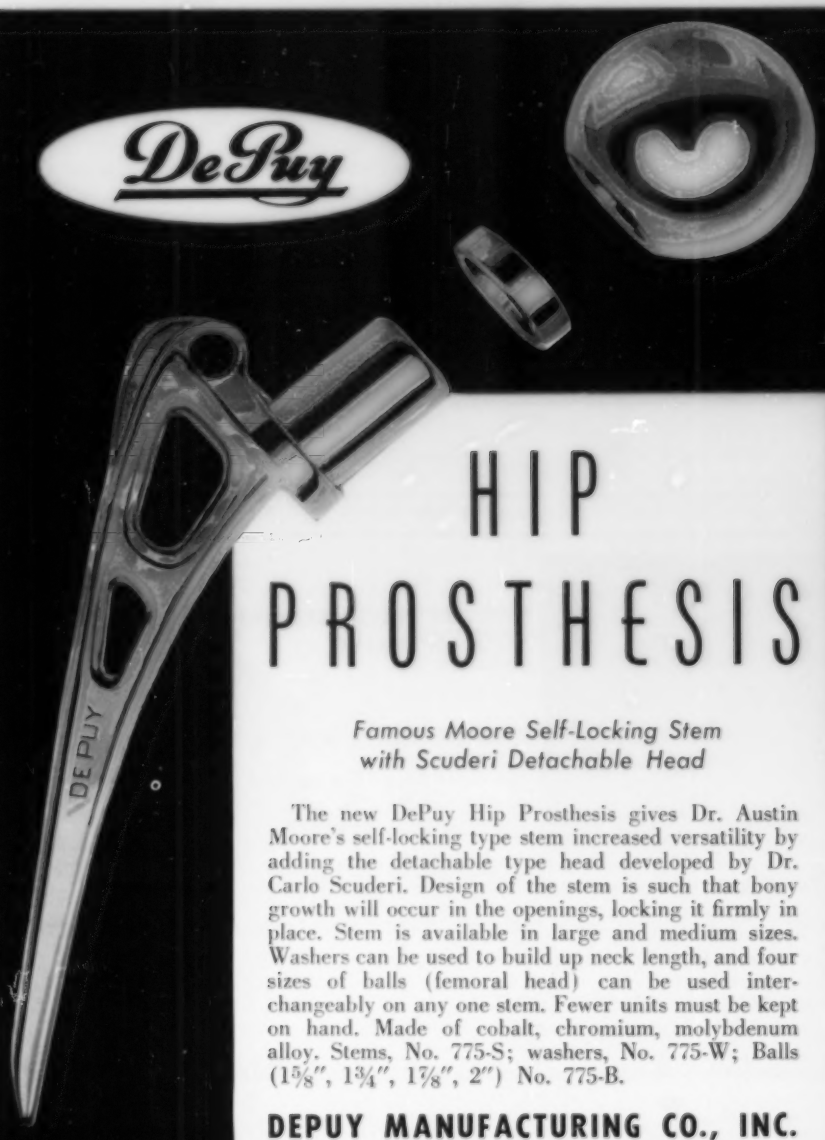
ORIGINATORS OF TARSO PRONATOR® AND TARSO MEDIUS® SHOES

Tarso shoes have been in use for over twenty-five years.

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

NEW

DePuy



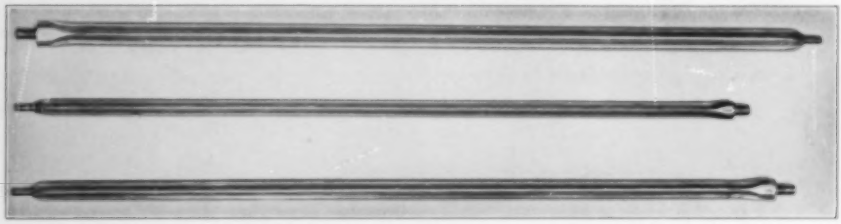
HIP PROSTHESIS

Famous Moore Self-Locking Stem with Scuderi Detachable Head

The new DePuy Hip Prosthesis gives Dr. Austin Moore's self-locking type stem increased versatility by adding the detachable type head developed by Dr. Carlo Scuderi. Design of the stem is such that bony growth will occur in the openings, locking it firmly in place. Stem is available in large and medium sizes. Washers can be used to build up neck length, and four sizes of balls (femoral head) can be used interchangeably on any one stem. Fewer units must be kept on hand. Made of cobalt, chromium, molybdenum alloy. Stems, No. 775-S; washers, No. 775-W; Balls (1⅝", 1¾", 1⅞", 2") No. 775-B.

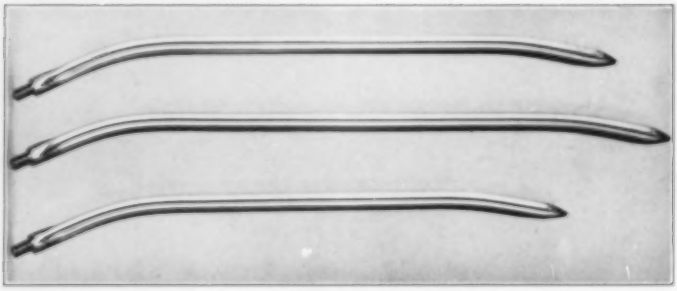
DEPUY MANUFACTURING CO., INC.
WARSAW, INDIANA
SINCE 1895—STANDARD OF QUALITY

LOTTES NAILS and INSTRUMENTS



***LOTTES* FEMORAL NAILS** are manufactured with diameters of ⅜, 7/16, and ½ inches —with lengths in ½ inch variations from 14 to 20 inches.

***LOTTES* TIBIAL NAILS** are made in 5/16 and ⅜ inch diameters—in lengths from 9 to 15½ inches, in variation of ½ inch.

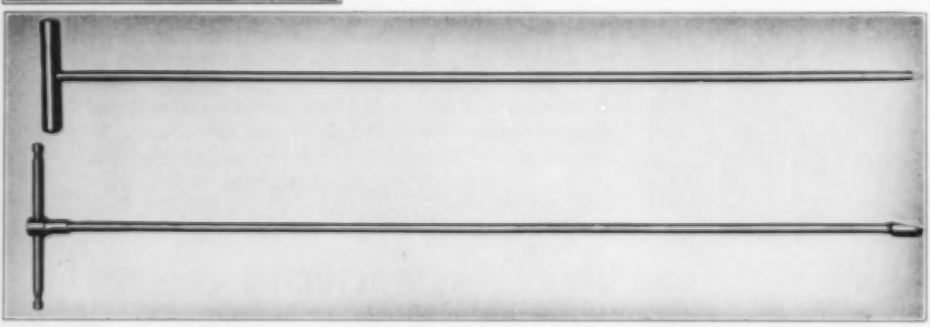


***LOTTES* NAILS AND INSTRUMENTS ARE MADE OF 18-8 SMo STAINLESS STEEL**

***LOTTES* DRIVER-EXTRACTOR COMBINATION and *LOTTES* MALLET**

***LOTTES* GUIDE WIRE AND *LOTTES* REAMER**

The reamer comes in 3 diameters: ⅜, 7/16, and ½ inch—all are 23 inches in length.



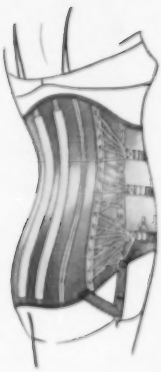
CHAS. A. SCHMIDT SURGICAL INSTRUMENT COMPANY

3689 Olive Street, Saint Louis 8, Missouri

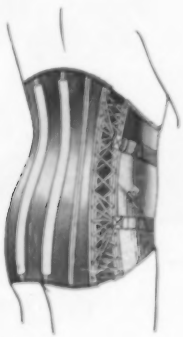
In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

FOR
ORTHOPEDIC
APPLICATIONS

Freeman SURGICAL SUPPORTS



MODEL 423
Sacro-Lumbar Back
Support for Women



MODEL 422
Sacro-Lumbar Back
Support For Men

For 68 years Freeman has worked closely with orthopedic surgeons to design surgical garments that provide *correct* support. Each garment is made by skilled craftsmen using the finest materials available.

There's a quality Freeman support for most every application. You can be certain that the garment you select will carry out your prescription to the best possible satisfaction of you and your patient.

Freeman corset-type back supports provide bracing in any desired degree to almost complete immobilization. Special construction features assure comfort for the wearer in sitting, standing and reclining positions.

FREEMAN QUALITY FEATURES

New Dacron-Pima Cotton Fabric. Petal-Soft Interior Finish cushions the stays, avoids irritation. Self-Smoothing, Exclusive Non-Wrinkle Fly speeds putting on garment and avoids comfort-robbing wrinkles. Soft Plush Padding under hooks and eyes. Superb Needle Work.

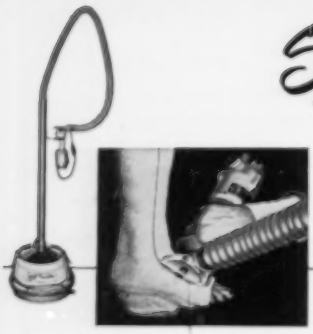
Prescribe **FREEMAN** *with Confidence!*

FREEMAN MANUFACTURING CO.
Dept. 701, Sturgis, Michigan
Please send details on new Freeman features and include free reference catalog.

Name_____

Address_____

City_____ Zone____ State_____



Today's concept in cast removal . . .

Stryker CAST CUTTER with PLASTERVAC

The Stryker Cast Cutter is universally accepted today as the most modern method for quick safe cast removal. The PlasterVac, when attached to the Cast Cutter, instantly picks up all dust and dirt as the cast is cut.

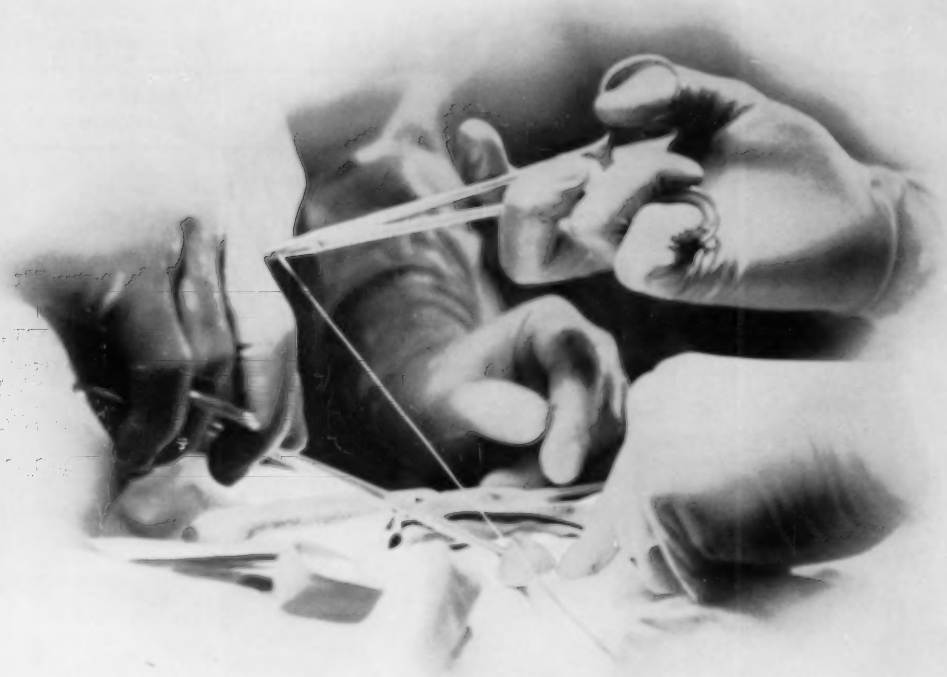
Write for additional information today.

SURGICAL AND HOSPITAL EQUIPMENT

Orthopedic Frame Company

420 ALCOTT STREET • KALAMAZOO, MICHIGAN

In answering advertisements, please mention *The Journal of Bone and Joint Surgery.*



Premedication *without* respiratory or circulatory depression

...and *with* 5 other advantages

PHENERGAN offers unmistakable aid to surgeon, anesthesiologist, and nurse. Proved in many thousands of patients, premedication with PHENERGAN curbs fear and excitement, prevents nausea and vomiting, facilitates anesthesia, reduces the requirement for depressant anesthetic and analgesic agents, and counteracts sensitivity reactions. All this without producing depression of the vital functions.

PHENERGAN® HYDROCHLORIDE

Promethazine Hydrochloride, Wyeth

INJECTION
TABLETS
SYRUP
SUPPOSITORIES

Wyeth®

Philadelphia 1, Pa.

New...conservative treatment for muscle and joint disease

- potent...fast relief in acute conditions
- safe...even for prolonged use in chronic cases

low back pain

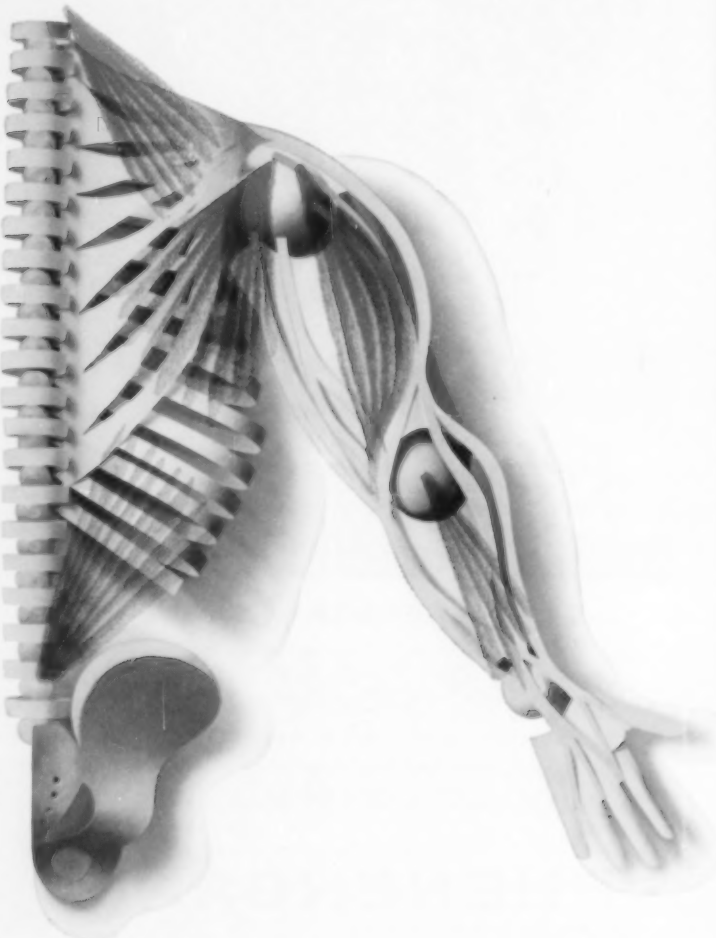
bursitis

strains and sprains

traumatic conditions

arthritis

myalgias



In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

SOMA RELIEVES PAIN in a unique way by modifying central perception of pain without abolishing natural defense reflexes.

SOMA RELAXES MUSCLE SPASM . . . approximately 8 times more potent than meprobamate or mephenesin.

PHYSICIANS' REPORTS:

"Marked pain-relieving effects of the new drug (SOMA) were seen in conditions involving muscle spasm and stiffness, whether acute or chronic. Relief from pain was usually rapid and sometimes dramatic." (90 patients.) Kuge, T.: To be published.

"In 86 per cent of the patients there were excellent or good results. . . . Relief of pain was noted by the patients' statements, by the diminished need for analgesic drugs, and by improved sleep." (154 patients.) Wein, A.B.: The Use of Carisoprodol in Orthopedic Surgery and Rehabilitation. Proceedings of the Symposium on the Pharmacology and Clinical Usefulness of Carisoprodol. Wayne State University Press, Detroit, 1959, p. 156.

In a double-blind study, SOMA was reported to be "clinically effective to a highly significant degree." (92 patients.) Cooper, C. D., and Epstein, J. H.: The Clinical Evaluation of Carisoprodol by a double-blind technique. *Ibid.* p. 97

Notable safety—extremely low toxicity; no known contraindications; side effects are rare; drowsiness may occur, usually at higher dosage

Fast action—starts to act promptly

Sustained effect—relief lasts up to 6 hours

Easy to use—usual adult dose is one 350 mg. tablet 3 times daily and at bedtime

Supplied—as white, coated 350 mg. tablets, bottles of 50.
Also available for pediatric use: 250 mg. orange capsules, bottles of 50.

SOMA™

(carisoprodol Wallace)

The only drug combining analgesia with muscle relaxation in a single molecule

BIBLIOGRAPHY: 1. Berger, F. M., Kletzkin, M., Ludwig, B. J., Margolin, S. and Powell, L. S.: J. Pharm. Exp. Ther. *127*:66, (Sept.) 1959. 2. Leake, Chauncey D.: Proceedings of the Symposium on The Pharmacology and Clinical Usefulness of Carisoprodol, Wayne State University Press. Detroit, 1959. p. 8. 3. Kestler, Otto: Ibid. p. 143. 4. Proctor, Richard C.: Ibid. p. 122. 5. Berger, Frank M.: Ibid. p. 25. 6. Goodgold, Joseph, Hohmann, Thomas and Tajima, Toshihiro: Ibid. p. 66. 7. Gammon, George D. and Tucker, Samuel: Ibid. p. 70. 8. Baird, Henry W. and Menta, Dominic A.: Ibid. p. 85. 9. Cooper, C. David and Epstein, Jerome H.: Ibid. p. 97. 10. Korst, Donald R., Gerard, R. W., Miller, James G., Small, Iver F., Graham, I. J. and Winkelman, Eugene I.: Ibid. p. 104. 11. Friedman, Arnold P.: Ibid. p. 115. 12. Trimpi, Howard D.: Ibid. p. 150. 13. Wein, Arthur B.: Ibid. p. 156. 14. Olds, James and Travis, R. P.: Ibid. p. 39. 15. Hess, Eckhard H., Polt, James M. and Goodwin, Elizabeth: Ibid. p. 51. 16. Phelps, Winthrop M.: Ibid. p. 131. 17. Spears, Catherine E.: Ibid. p. 138. 18. Hyde, L. P. and Hough, Charles E.: Ibid. p. 166. 19. Spears, Catherine E. and Phelps, Winthrop M.: Arch Pediat., 76:287 (July) 1959. 20. Phelps, Winthrop M.: Arch. Pediat., 76:243, (June) 1959. 21. Friedman, Arnold P.: Paper presented at Scientific Meeting, New York State Society of Industrial Medicine, Inc., New York, Sept. 30, 1959. 22. Frankel, Kalman: Ibid. 23. Fransway, Robert L.: Ibid. 24. Kuge, T.: Unpublished reports.

Literature and samples on request. WALLACE LABORATORIES, New Brunswick, New Jersey

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

STRAIGHT LAST

REGULAR

ARCH FEATURE

MISMATES

You'll find everything you want and need in

Child Life SHOES

a complete line

Here are shoes for children that adapt themselves readily to your corrective prescriptions . . . yet every shoe is styled to look like a "regular" shoe . . . freeing your patients from any inferiority complex that might develop from having to wear shoes that are "different" in appearance from those worn by other children.

For over 35 years the makers of CHILD LIFE shoes have concentrated exclusively on juvenile footwear . . . making only quality shoes of leather for both "regular" wear and orthopedic service. Nine chances out of ten there is a specially selected CHILD LIFE dealer in your community who will be glad to accept your prescription work and who is competent to handle your instructions. Write for his name, and for the CHILD LIFE service booklet for the professional man, featuring representative shoes and wedging procedures.

HERBST Shoe Manufacturing Company
Box 2005 Milwaukee 1, Wisconsin

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

meperidine-promethazine combined

MEPERGAN®

Promethazine Hydrochloride and Meperidine Hydrochloride, Wyeth

MEPERGAN provides the analgesia of meperidine augmented by the values of promethazine. These include the allaying of apprehension, the control of nausea and vomiting, the potentiation of barbiturates and narcotics, and the facilitation of anesthesia. MEPERGAN is useful in all forms of surgery. It may be used preoperatively for preparatory medication, during surgery as an adjunct to anesthesia, and postoperatively to control pain. Widespread clinical experience points to MEPERGAN's 1:1 ratio of promethazine and meperidine to be most satisfactory for most patients.

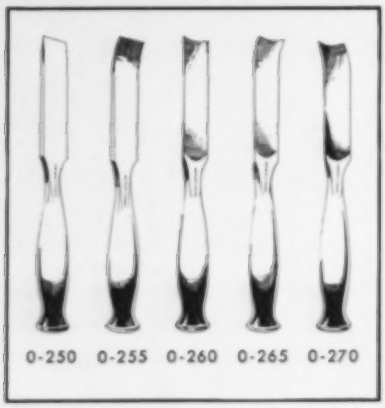
Wyeth Laboratories, Philadelphia 1, Pa.

See package circular for complete information on use of MEPERGAN.

100 ANNIVERSARY Wyeth

A Century of Service to Medicine

SMITH-PETERSEN OSTEOTOMES and GOUGES



0-250 0-255 0-260 0-265 0-270

0-250 SMITH-PETERSEN OSTEOTOME—straight, 8", sizes ¼", ⅜", ½", ⅝", ¾", 1" and 1¼". Stainless Steel.

0-255 SMITH-PETERSEN OSTEOTOME—curved, 8", sizes ¼", ⅜", ½", ⅝", ¾", 1" and 1¼". Stainless Steel.

0-260 SMITH-PETERSEN GOUGE—straight, 8", sizes ⅜", 9/16", ¾" and 1". Stainless Steel.

0-265 SMITH-PETERSEN GOUGE—curved, 8", sizes ⅜", 9/16", ¾" and 1". Stainless Steel.

0-270 SMITH-PETERSEN GOUGE—reverse curved, 8", sizes ⅜", 9/16", ¾" and 1". Stainless Steel.

A NEW STAINLESS STEEL CASE TO HOLD SMITH-PETERSEN OSTEOTOMES and GOUGES

0-295 Open

0-295 Closed

CASE 0-295—holds seven Smith-Petersen Osteotomes.

CASE 0-296—holds seven Smith-Petersen Gouges.

CASE 0-297—holds a combination of seven Osteotomes and Gouges.

Cutting edges are protected at all times. Complete unit may be autoclaved.

Specify Width of Gouges and Osteotomes When Ordering

ESTABLISHED 1858

Codman & Shurtleff, Inc.

MAKERS OF SURGEONS' INSTRUMENTS

104 BROOKLINE AVENUE BOSTON 15, MASSACHUSETTS

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

Spencer® SOLVES ANOTHER PATIENT'S TOTAL SUPPORT REQUIREMENTS

● Spencer first realigns the body — then designs a support to hold it that way.

Previous braces for this patient with scoliosis and kyphosis were cumbersome and conspicuous.

Her **Spencer** combines proper anatomical support with cosmetic acceptability.

She indicates Spencer is 100% better. She could not walk, sit or do her housework without it. It gives her better support and makes her stand taller as well as improving her figure and general well-being.

● Note unique placement of steels for maximum support and greatest tolerance.

Spencer's Individual Designing Service is available to you through Corsetieres specially trained to help you help your patients.

SPENCER®

individually designed supports for women, men and children

SPENCER, INCORPORATED
19 Ellsworth Ave., New Haven 7, Conn.
Canada: Spencer, Ltd., Rock Island, Quebec
England: Spencer, Ltd., Banbury, Oxon

☐ Send me the name of the nearest Spencer Corsetiere.

☐ I would like an office demonstration of Spencer benefits and features.

Name _____

Address _____

_____

1-60

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

Babies are bundles of activity. As they grow, they channel their energy into pulling, pushing, crawling, and finally, one triumphant day, they're walking. If it's one of 'your' babies, you're just as proud as parents when you see a child running on strong legs and sturdy feet.

Edwards is proud as parents, too, because so many children learn to run and romp in Edwards shoes. Edwards dealers carry the three types of shoes shown below for all kinds of growing feet.

Edwards®

Recommend and prescribe Edwards *shoes for 'your' babies. For more information, send for* Edwards *brochure and prescription blanks.*

The Shoe for Children, Philadelphia 7, Penna.

PEDIC

PRONA-PEDIC Edwards P P

Edwards Toddlins—for beginners, the tiny tots just learning to navigate.

Edwards Pedics—for children with foot disorders that you prescribe for.

Edwards Regulars — for normal healthy children's feet—with emphasis on proper fit.

PROVEN EFFECTIVE FOR THE TENSE AND NERVOUS PATIENT

"There is perhaps no other drug introduced in recent years which has had such a broad spectrum of clinical application as has meprobamate.* As a tranquilizer, without an autonomic component in its action, and with a minimum of side effects, meprobamate has met a clinical need in anxiety states and many organic diseases with a tension component."

Krantz, J. C., Jr.: The restless patient—A psychologic and pharmacologic viewpoint. Current M. Digest 25:68, Feb. 1958.

*Miltown®

the original meprobamate, discovered and introduced by

WALLACE LABORATORIES, *New Brunswick, N. J.*

CM-215

Louis C. Weld, President, G. W. Chesbrough Co. . . . "My own personal experience led to the development of Chesbrough Orthopedic Pre-Walkers, club-foot, open toe and closed toe Surgicals."

"Orthopedic shoes parents can afford . . .

CHESBROUGH Corrective Pre-Walkers"

"When a child in my own family needed a corrective shoe, I discovered what a strain it can mean to a family budget, because 1) corrective footwear is expensive and 2) frequent purchase of new corrective shoes is required. Then and there I decided there was a real need for a moderately priced corrective shoe —a shoe parents could afford. That's why and when Chesbrough Orthopedic Pre-Walkers were born.

"Our 60 years of shoe-making experience resulted in corrective Pre-Walkers of scientific design, expert workmanship, fine leathers combined with orthopedically correct lasts to provide necessary correction at an economical price."

Today—just three years later—orthopedic specialists in 45 states and several foreign countries are prescribing these shoes.

All shoes in unlined white elk, sizes 000 to 4, narrow and wide. Available in full pairs, split pairs or single shoes (no extra charge for half pairs).

No. 1400 OPEN TOE. Straight-line symmetrical last, firm heel, no back seam. Adaptable to Denis Browne Splints.

No. 1700 CLUBFOOT, OPEN TOE. Special outflare last, sturdy instep strap to stabilize heel.

No. 1300 CLOSED TOE. Lace-to-toe design permits snug, gentle fit. Perfectly smooth inside.

MAIL COUPON FOR FREE DESK SAMPLES

G. W. CHESBROUGH CO.
807 Smith Street, Rochester 6, N. Y.

NAME.....

ADDRESS.....

CITY.....

YOUR ORTHOPEDIC SHOE DEALER:

.....

SMo

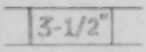
Type 316 *Stainless Steel*

IMPLANTS

The material of Wright SMo (Type 316) Stainless Steel Implants is INERT and exists on the friendliest terms with bone, body fluids and soft tissue. All Wright Implants are Xygio Pentrex tested and inspected and are electro-polished and packed in polyethylene envelopes to assure the surgeon of complete metal surface cleanliness. All Wright Products are unconditionally guaranteed against defective workmanship and materials.

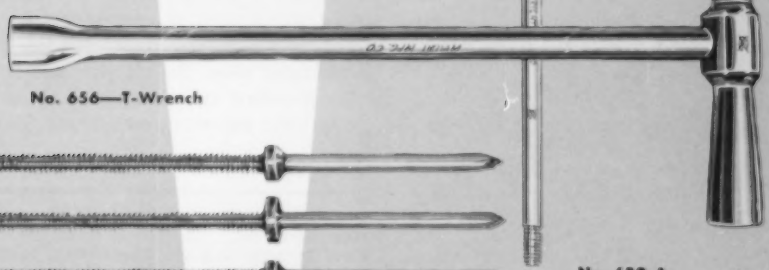
No. 155—Threaded Pin

No. 223—Knowles Pin

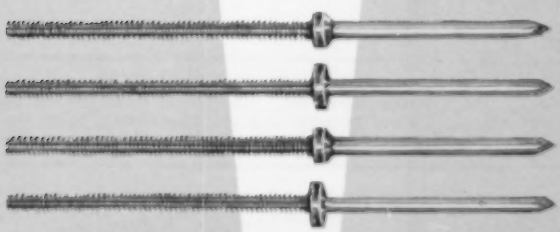


SIZES MARKED ON EACH PIN

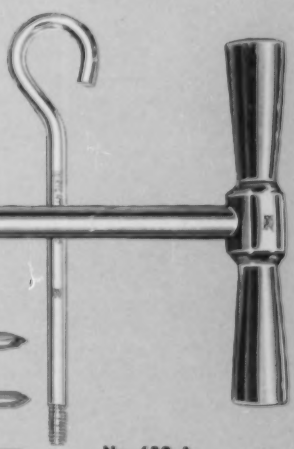
No. 680—R. Beverley Ray Lateral Traction Screw



No. 656—T-Wrench



No. 222 Moore Pins *Set of Four*



No. 680-A Traction Hook

No. 224-SP— Jewett Hip Nail with Slotted Plate

Wright MANUFACTURING COMPANY

880, 882 ADAMS • MEMPHIS 3, TENNESSEE

Write for further information and new catalog to Dept. O-16

Puts new spring in your step

Spring-action

*RIPPLE® Sole SHOES

The SURGEON
Tie-style, arch counter.
$21.95** ppd.

The NURSE
Tie-style, arch counter.
$20.95** ppd.

* TM Ripple Sole Corp.

Here's good news for those who stand or walk long hours on their feet . . . a new, super-flexible "footing", RIPPLE® Sole, called in an article in Reader's Digest: "the most important improvement in footwear since Julius Caesar put heels on the boots of his legionnaires".

A series of flexible corrugations that depress when stepped on, then spring back to put pep in your step, RIPPLE® Sole has been tested and proven to help absorb shock, cause of most foot fatigue. Many doctors and nurses wear them, "live" in them. Try a pair yourself. Take a few steps. You'll never wear a more comfortable shoe. Satisfaction guaranteed. ORDER TODAY (give size, width) or write for FREE folder (smart styles for the whole family). **Mich. orders add 3% tax. (Men's, over size 12, add $1.50; women's over 10, add $1.) Conductive plug and heel seat, $3 extra.

Let shock-absorbing RIPPLE® Sole shoes ease every step for your patients as well. Remember . . . for perfection of fit . . . they can count on the Hack Shoe Co.'s 43-year reputation . . . and because only the finest-fitting footwear has ever carried a Hack label, you can recommend them with confidence.

THE HACK SHOE COMPANY, 504 Mutual Building, Detroit 26, Michigan

Please send me ☐ Technical information on RIPPLE® Sole
☐ Free style folder ☐ NURSE ☐ SURGEON
Size....Width....

NAME_____

ADDRESS_____

CITY_____ ZONE_____ STATE_____

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

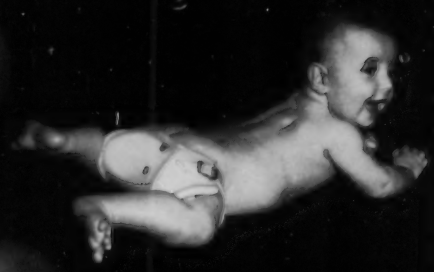
Richards

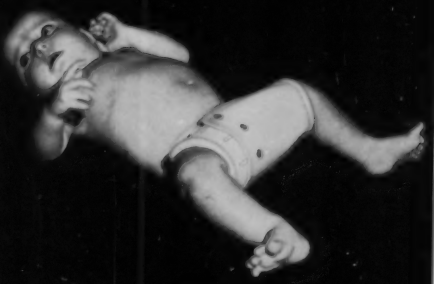
CRAIG ADJUSTABLE HIP SPLINT

(Patent Pending)

For the treatment of:

CONGENITAL DYSPLASIA AND DISLOCATION OF THE HIP





In the treatment of congenital dysplasia and dislocation of the hip under two years, an ideal splinting position is with the hips at 90° flexion and 90° abduction.

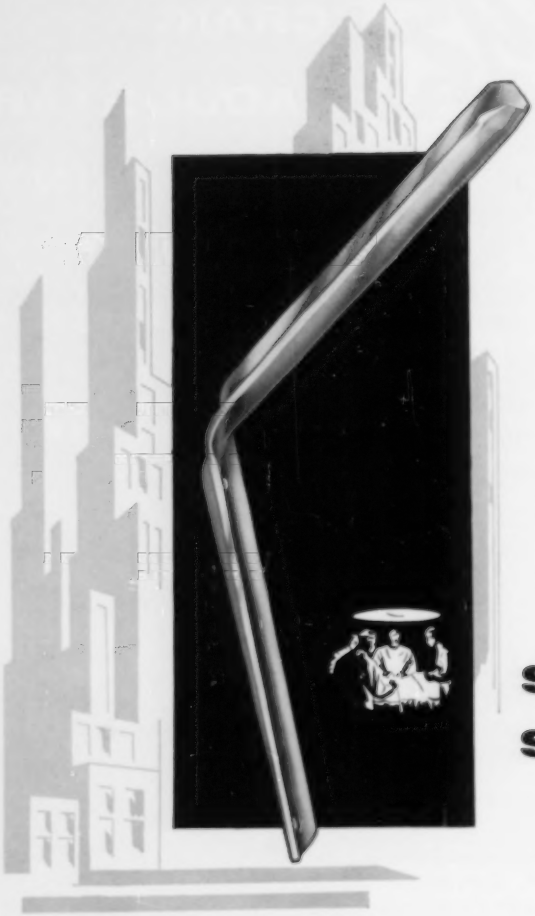
Tubular shaped when on the patient, the Craig Adjustable Hip Splint holds the hips comfortably in this correct position without rigid or bulky immobilization. It is impervious to water, thus completely sanitary. Constructed of frosty white plastic with plastic covered foam rubber binding to prevent irritation. The splint retains the diapers, eliminating the need for safety pins. Two snap-button fasteners makes diaper changing easy.

By a simple "button-hole" adjustment, the splint is suitable for all infants from birth to two and one-half years.

WRITE FOR FULL INFORMATION

Richards MANUFACTURING CO.

756 Madison Avenue, Memphis, Tennessee



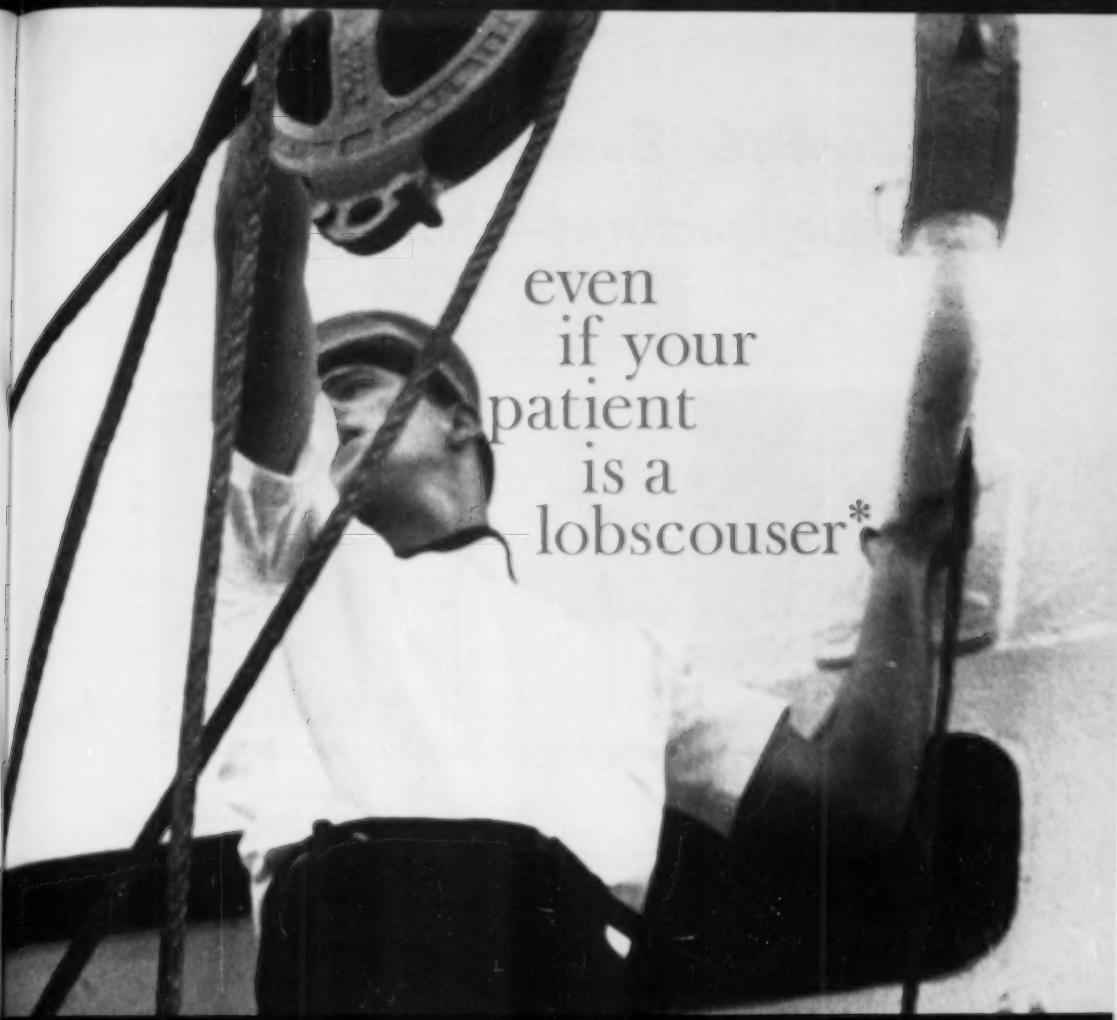
STRUCTURALLY SOUND

NEUFELD FEMORAL NAIL PLATES
have been engineered to provide maximum strength at the bend. Available in either Neutrilium (cobalt alloy) or type 316 stainless steel with screws of identical metals . . . The Neufeld nail is the choice of careful, discriminating orthopedic surgeons.

Ask your Surgical Supply Dealer or
write for additional technical information

MEDICAL RESEARCH SPECIALTIES
Loma Linda, California

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.



he'll be under way again soon, once he's on

PARAFON®

(PARAFLEX® + TYLENOL®)

for muscle relaxation plus analgesia

prescribe PARAFON in low back pain—sprains—strains—rheumatic pains

Each PARAFON tablet contains:

PARAFLEX® Chlorzoxazone† ..... 125 mg.

Specific for skeletal muscle spasm

TYLENOL® Acetaminophen ..... 300 mg.

The analgesic preferred in musculoskeletal pain

Dosage: Two tablets t.i.d. or q.i.d.

Supplied: Tablets, scored, pink, bottles of 50.

and in arthritis

PARAFON®

with Prednisolone

Each PARAFON WITH PREDNISOLONE tablet contains: PARAFLEX® Chlorzoxazone† 125 mg., TYLENOL® Acetaminophen 300 mg., and prednisolone 1.0 mg.

Dosage: One or two tablets t.i.d. or q.i.d.

Supplied: Tablets, scored, buff colored, bottles of 36.

Precautions: The precautions and contraindications that apply to all steroids should be kept in mind when prescribing PARAFON WITH PREDNISOLONE.

*sailor

†U. S. Patent Pending

McNEIL

McNeil Laboratories, Inc • Philadelphia 32, Pa.

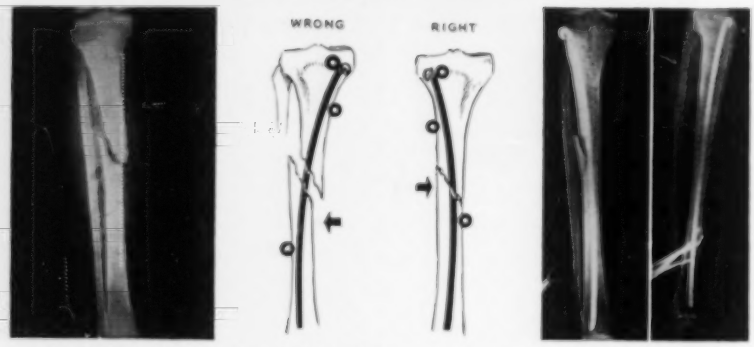
254459

Lock the Bone with Dynamics

Oblique Fractures—Short Fragments*

The RUSH® MEDULLARY PIN

PAT. NO. 2,579,968



Direction of pin insertion determines compression or distraction.

V (victory) =
Good Compression

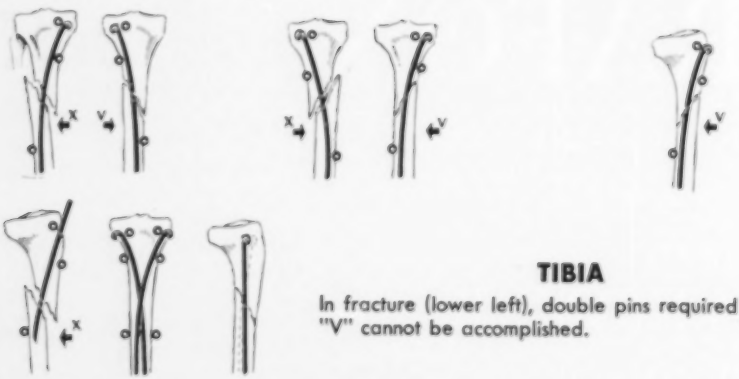
X (no good) =
Distraction

RADIUS

Length of fragment and direction of fracture line determine angle of pin insertion.

RULE

Axis of pin should be same direction as axis of fracture line, intersecting to make a "V" instead of an "X".

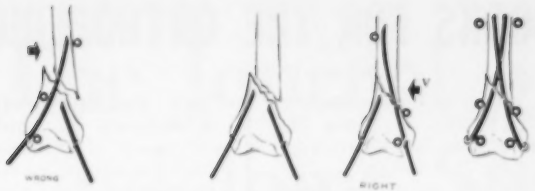


TIBIA

In fracture (lower left), double pins required because "V" cannot be accomplished.

* Rush, Leslie V.; Fracture Problems, Mississippi Doctor, Vol. 36, No. 10, March 1959.

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

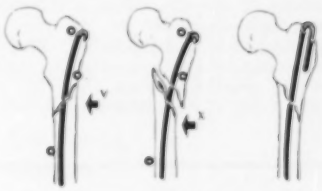


LOWER FEMUR

Double pins required. Introduce most effective pin first.

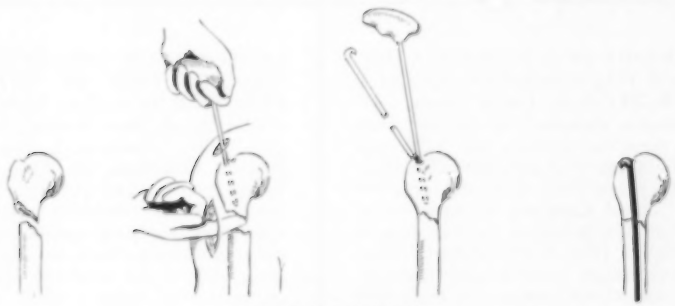
UPPER FEMUR

Dependent upon direction of fracture line, special type pin might be indicated.



HUMERUS, BELOW DELTOID

Requires precise handling for good healing. For fracture at right use large curved pin for compression.



UPPER THIRD

To avoid angulation, insert pin axially in short fragment.

WRITE FOR INFORMATION
Exclusive Manufacturers of
RUSH MEDULLARY PINS AND INSTRUMENTS
Publishers of
"ATLAS OF RUSH PIN TECHNICS"
by LESLIE V. RUSH, M. D.

THE BERIVON company
MERIDIAN, MISS.

6 NEW BOOKS FOR THE ORTHOPEDIC SURGEON

PROSTHETIC PRINCIPLES — ABOVE KNEE AMPUTATIONS *by* **Miles H. Anderson, John J. Bray,** and **Charles A. Hennessy,** *all of the University of California School of Medicine.* Edited by **Raymond E. Sollars.** Offering COMPLETE COVERAGE of all matters relating to the fitting and fabrication of the above-knee prosthesis, beginning with functional anatomy of the hip, thigh, and knee and a thorough discussion of the principles of locomotion — and proceeding through complete step-by-step procedure for fabrication of the prosthesis. *Pub. Dec. '59, 348 pp. (8½ x 11), 677 il., $10.00*

THE SURGICAL TREATMENT OF SCOLIOSIS *by* **Louis A. Goldstein,** *University of Rochester Medical Center.* Based on personal experiences of the author in the surgical treatment of scoliosis over a twenty-year period — all cases treated by one method and followed up by Doctor Goldstein. ALL VARIABLES ELIMINATED. Thirty-one cases illustrated. Preoperative and postoperative roentgenograms and clinical photographs emphasize important points in the discussion of TREATMENT and END RESULTS. *Pub. Oct. '59, 128 pp., 265 il. (Amer. Lec. Orthopedic Surgery), $6.75*

ATLAS OF ANATOMY AND SURGICAL APPROACHES IN ORTHOPAEDIC SURGERY *by* **Rodolfo Cosentino,** *University of La Plata, Argentina.* Nothing like these illustrations has heretofore appeared in anatomical or orthopedic surgery literature. Demonstrating with far greater accuracy the anatomical structures encountered by the operator from layer to layer, the author's FRESH AND VIGOROUS ABILITIES IN DISSECTION AND IN DESCRIPTIVE ANATOMY make his approaches indispensable to the road which counts most in orthopedic surgery. *Publication date February 1960*

CRASH INJURIES: THE INTEGRATED MEDICAL ASPECTS OF AUTOMOBILE INJURIES AND DEATHS *by* **Jacob Kulowski,** *St. Joseph, Missouri.* The WHY and HOW of automobile accidents and injuries. A large part of the book is on HUMAN SALVAGE — from first aid through ultimate rehabilitation. Includes all available scientific and empiric knowledge of today which the physician needs to meet the challenge of PATIENT TOTAL CARE derived from traffic and other injuries of mechanical origin. Over 50 tables and 600 illustrations serve to clarify the situation. *Publication date March 1960*

FELLOWSHIP OF SURGEONS: A HISTORY OF THE AMERICAN COLLEGE OF SURGEONS *by* **Loyal Davis,** *Chicago, Illinois.* Revealed for the first time are the thoughts, motives, and actions of legendary figures of surgery and allied medical and hospital fields from the inception of the Clinical Congress of Surgeons of North America, including the founding of the College in 1913, to the present. Taken from unpublished archives and from recorded personal reminiscences of those who were on the scene in the early days, the History is a FASCINATING ACCOUNT OF THE SUCCESSFUL STRUGGLE TO ELEVATE THE STANDARDS OF PATIENT CARE. *Publication date February 1960*

LECTURES ON THE INTERPRETATION OF PAIN IN ORTHOPEDIC PRACTICE *by* **Arthur Steindler,** *State University of Iowa Medical School, Iowa City.* With Anatomical Dissections by Rodolfo Cosentino, *La Plata, Argentina.* In his present and possibly his greatest work, Doctor Steindler analyzes pain syndromes applying specifically to orthopedic situations. Such an analysis means recognition of the condition while it is still in reversible stage — while appropriate measures of prevention can be taken. Sit in with Doctor Steindler as he shows you HOW TO FACILITATE EARLIER DIAGNOSIS AND MORE EFFECTIVE MANAGEMENT of conditions involving the locomotor apparatus. *Pub. Oct. '59, 680 pp., 334 il., $18.50*

CHARLES C THOMAS · PUBLISHER 301-327 East Lawrence Avenue **SPRINGFIELD · ILLINOIS**

The Journal of Bone and Joint Surgery

The Official Publication of

The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association
American Society for Surgery of the Hand
New Zealand Orthopaedic Association
South African Orthopaedic Association

AMERICAN VOLUME

VOL. 42-A

8 THE FENWAY, BOSTON 15, MASSACHUSETTS, U. S. A.

BRITISH OFFICE:
82 PORTLAND PLACE, LONDON, W. 1, ENGLAND

The Journal of Bone and Joint Surgery

PUBLISHED IN TWO VOLUMES

WILLIAM A. ROGERS, *Editor-in-Chief Emeritus*

AMERICAN VOLUME

Editor and Chairman Board of Editors: THORNTON BROWN

American Board of Associate Editors

CHARLES O. BECHTOL, Los Angeles, California
WILLIAM H. BICKEL, Rochester, Minnesota
DAVID M. BOSWORTH, New York, N. Y.
CRAWFORD J. CAMPBELL, Albany, New York
JONATHAN COHEN, Boston, Massachusetts
ALBERT B. FERGUSON, JR., Pittsburgh, Pennsylvania
S. BENJAMIN FOWLER, Nashville, Tennessee
CHARLES H. FRANTZ, Grand Rapids, Michigan
J. LEONARD GOLDNER, Durham, North Carolina
DAVID S. GRICE, Philadelphia, Pennsylvania
GEORGE HAMMOND, Boston, Massachusetts
CHARLES H. HERNDON, Cleveland, Ohio
FLOYD H. JERGESEN, San Francisco, California
WILLIAM A. LARMON, Chicago, Illinois
LEO S. LUCAS, Portland, Oregon
MICHAEL L. MASON, Chicago, Illinois
JOHN H. MOE, Minneapolis, Minnesota
JOHN ROYAL MOORE, Philadelphia, Pennsylvania
JESSE T. NICHOLSON, Philadelphia, Pennsylvania
FRED C. REYNOLDS, St. Louis, Missouri
DANIEL C. RIORDAN, New Orleans, Louisiana
LEE RAMSAY STRAUB, New York, N. Y.
VERNON P. THOMPSON, North Hollywood, California

BRITISH VOLUME

SIR REGINALD WATSON-JONES, *Editor*

British Editorial Board

SIR WALTER MERCER, *Chairman*
H. JACKSON BURROWS, *Deputy Editor*
J. CRAWFORD ADAMS, *Assistant Editor*
W. D. COLTART, *Treasurer*
C. E. L. ALLEN, Cape Town, South Africa
ROLAND BARNES, Glasgow, Scotland
J. S. BATCHELOR, London, England
JOHN CHARNLEY, Manchester, England
F. P. DEWAR, Toronto, Canada
J. KENNEDY ELLIOTT, Wellington, New Zealand
A. L. EYRE-BROOK, Bristol, England
T. J. FAIRBANK, Cambridge, England
ROGER GARIÉPY, Montreal, Canada
B. KEON-COHEN, Melbourne, Australia
DOUGLAS PARKER, Hobart, Tasmania
R. G. PULVERTAFT, Derby, England
ROBERT ROAF, Oswestry, England
H. J. SEDDON, London, England
E. W. SOMERVILLE, Oxford, England
GORONWY THOMAS, Liverpool, England
O. J. VAUGHAN-JACKSON, London, England

Corresponding Editors

Argentina: JOSÉ VALLS
Austria: WALTHER EHALT
Belgium: JEAN VERBRUGGE
Brazil: F. E. GODOY MOREIRA
Cuba: ALBERTO INCLÁN
Denmark: JOHANNES MORTENS
Finland: K. E. KALLIO
France: R. MERLE D'AUBIGNÉ
Germany: MATHIAS HACKENBROCH
Italy: O. SCAGLIETTI
Japan: EISHI KONDO
Mexico: JUAN FARILL
Netherlands: J. D. MULDER
Poland: ADAM GRUCA
Portugal: ARNALDO RODO
Sweden: STEN FRIBERG
Uruguay: JOSÉ LUIS BADO

Committees

THE JOURNAL OF BONE AND JOINT SURGERY, INCORPORATED

The American Orthopaedic Association

The American Academy of Orthopaedic Surgeons:

BOARD OF TRUSTEES

DAVID M. BOSWORTH, *Chairman*
KEENE O. HALDEMAN, *Vice-Chairman*
EDWARD L. COMPERE, *Secretary*
H. RELTON MCCARROLL, *Treasurer*
THORNTON BROWN, *Editor*
JAMES S. SPEED
GUY A. CALDWELL

American Society for Surgery of the Hand

President: JULIAN M. BRUNER
President-Elect: DANIEL C. RIORDAN
Secretary-Treasurer: GEORGE S. PHALEN
2020 East 93rd Street, Cleveland 6, Ohio

The Australian Orthopaedic Association

JOHN LAHZ, Brisbane, *President*
WARWICK STENING, Sydney, *Editorial Secretary*
RICHARD HODGKINSON, *Secretary*, 135 Macquarie Street, Sydney, New South Wales

The British Orthopaedic Association

H. J. SEDDON, *President*
SIR REGINALD WATSON-JONES, *Editor*
E. W. SOMERVILLE, *Editorial Secretary*
J. CRAWFORD ADAMS, *Secretary*
47 Lincoln's Inn Fields, London, W.C. 2

The Canadian Orthopaedic Association

W. B. MACKINNON, Winnipeg, *President*
W. W. OLLERHEAD, London, *Vice-President*
IAN W. DAVIDSON, *Secretary-Treasurer*
130 Elm Street, Sudbury, Ontario
F. P. DEWAR, *Editorial Secretary*
706 Medical Arts Building, Toronto, Ontario

New Zealand Orthopaedic Association

H. WALDEN FITZGERALD, Dunedin, *President*
ALAN ALLRED, Dunedin, *Secretary-Treasurer*
N. NISBET, *Editorial Secretary*
Dunedin Hospital, Dunedin, C.1, New Zealand

South African Orthopaedic Association

A. J. HELFET, *President*
SIDNEY SACKS, *Treasurer*
GEORGE DOMMISSE, *Secretary*
303 van Riebeek Medical Buildings, Pretoria, South Africa

Published in the United States by The Journal of Bone and Joint Surgery, Incorporated

Copyrighted in the United States by The Journal of Bone and Joint Surgery, Incorporated

Published in the United Kingdom for The British Editorial Society of Bone & Joint Surgery by E. & S. Livingstone, Teviot Place, Edinburgh

Copyright in Britain and the Commonwealth by The British Editorial Society of Bone & Joint Surgery

THE JOURNAL OF BONE AND JOINT SURGERY

American Office

8 The Fenway, Boston 15, Massachusetts, U.S.A.
Managing Editor: John A. Reed
Assistant Editor: H. Stephen Tilton

British Office

82 Portland Place, London, W. 1, England
Editorial Secretary: Jean Barnes

VOLUME 42-A, NO. 1 JANUARY 1960

The Journal of
Bone and Joint Surgery

American Volume

Congenital Absence of the Fibula*

BY A. W. FARMER, M.D., AND C. A. LAURIN, M.D., TORONTO, ONTARIO, CANADA

From the Department of Surgery, Hospital for Sick Children, Toronto

This is a review of thirty-two limbs with congenital absence of the fibula in twenty-four patients seen at the Hospital for Sick Children, Toronto. The etiology, pathology, diagnosis, and treatment of the anomaly will be discussed.

ETIOLOGY

The cause of congenital anomalies remains unknown, but genetic and environmental factors are generally acknowledged. In congenital absence of the fibula the responsible agents must be active before the eighth week of fetal life, as the limb buds usually appear in the sixth or seventh week [11]. No genetic factor has ever been satisfactorily demonstrated. Experimental teratogenesis, however, has supplied ample evidence that environmental forces may be a factor in the production of congenital deformities. Absence of the fibula and other limb anomalies have been experimentally produced by diet alterations [11], x-ray radiation [1], and inoculations of insulin [6].

The statements just made are compatible with the "archipterygium" theory of Gegenbaur, which suggests no specific etiological agent, but helps to explain the distribution of the associated anomalies.

PATHOLOGICAL ANATOMY

The presence of various anomalies in a limb with congenital absence of the fibula suggests that there is dysplasia of the whole limb. The appearance of the limb is usually as follows: the limb is short, the tibia is bowed, and the foot is deformed in an equinovalgus position (Figs. 10-A and 10-B).

Most of the shortening of the limb is due to the short, curved tibia, but shortening of the femur is also usually present, although to a lesser degree (Fig. 1). Occasionally, there may be almost complete absence of the femur. When the absence of the femur is bilateral, the tibiae are usually straight (Fig. 2-B).

The predominant bowing of the tibia is anterior, with thickening of the cortex on the concave side (Figs. 3-A and 3-B). Medial bowing is not so common, but it occurs in the more severe cases. When present, it contributes to the distorted position of the foot. The lower tibial epiphysis is frequently abnormal and is slow to ossify.

If the foot is deformed, as is frequently the case, the big toe is usually present, but the lateral tarsal bones and toes are lacking. The calcaneus is in severe equinus position, and at times is displaced posterolateral to the lower end of the tibia. If the calcaneus is displaced in this way, the talus is medial and distal

*Read at the Joint Meeting of the Orthopaedic Associations of the English-Speaking World, Washington, D. C., May 12, 1958.

to it. Solid bone coalition of these two tarsal bones is a common occurrence. In some cases, even with a partial absence of the femur and the fibula, the foot is normal and the ankle joint functionally adequate (Fig. 2-A).

The absence of the fibula may be complete or incomplete as shown by roentgenography (Figs. 4-A through 4-C). With incomplete absence, the ossification may be considerably delayed, and early roentgenograms may be misleading.

The peroneal tendons, the Achilles tendon, the posterolateral ligaments, and the capsule of the ankle and subtalar joints are shortened and contribute to the deformity.

FIG. 1

Shortening of the limb due to dysplasia of both the tibia and the femur. Note the ipsilateral dysplasia of the hip.

DIAGNOSIS

The diagnosis is straightforward in the newborn infant, as only two other congenital lesions need to be considered: congenital bowing of the tibia, in which the fibula is present and the foot is normal (Figs. 5-A through 5-C); and congenital absence of the tibia in which the foot is in varus position, the knee is dislocated, and there is little bowing of the fibula (Figs. 6-A through 6-C).

REVIEW OF CASES

Thirty-two limbs with congenital absence of the fibula in twenty-four patients were seen at the Hospital for Sick Children, Toronto, from 1931 to 1957. Congenital absence of other long bones was not so common, for, during that same interval, complete or incomplete absence of the following long bones was noted: the femur in sixteen patients; the radius in thirteen patients; the tibia in five patients; and the ulna in two patients. Excluding cases of complete congenital

FIG. 2-A

FIG. 2-B

Fig. 2-A: With unilateral absence of the fibula associated with absence of the femur there is a severe discrepancy in the length of the limbs. Note that even with severe shortening due to dysplasia of the femur and tibia, the foot is still functionally adequate in this case. A modified Syme amputation is often the best solution in the unilateral case, since it permits the use of a simple end-bearing similar prosthesis. Note that there is no bowing of the tibia.

Fig. 2-B: When the deformity is bilateral, a discrepancy in the length of the limbs is less often a problem.

FIG. 3-A

FIG. 3-B

Tibial bowing may regress with advancing age. Note the change of curvature from that at the age of three months to that at the age of three years. Note also the thickening of the tibial cortex on the concavity of the curve.

FIG. 4-B

FIG. 4-C

Figs. 4-A through 4-C: Varying degrees of absence of the fibula.

Fig. 4-A (upper left): Calcification of the distal end of the fibular remnant.

Fig. 4-B: The proximal end of the fibula is absent.

Fig. 4-C: There is complete absence of the fibula on the left and delayed ossification of the proximal fibular epiphysis on the right. Note the abnormal lower tibial epiphysis and the talocalcaneal coalition on the side of the complete absence of the fibula.

amputation and phocomelia, there were no cases of absence of the humerus. These statistics closely reflect the relative incidence of these anomalies.

In most cases of micromelia, shortening is rarely restricted to one bone (Table I). In sixteen patients with unilateral absence of the fibula, shortening was always noted in the tibia. Thirteen of these patients also had shortening of the femur. In the dysplasia of the limb which accompanies congenital absence of the fibula, there is usually shortening of the femur as well as of the tibia.

In this series, the absence of the fibula was incomplete as shown on roentgenograms in five cases (Figs. 4-A through 4-C); the absent portion was always the distal end of the fibula. Similarly, with an incomplete absence of the femur the distal end was usually missing.

Anterior bowing and shortening of the tibia were noted in seventeen patients; in the severe cases medial bowing was also present. The tibia was straight in four patients who also had partial absence of the femur (Fig. 2-A) and in three patients with milder (Type I) deformities (Figs. 7-A, 7-B, and 7-C).

The foot was anatomically incomplete in sixteen patients. It was always in some valgus position and usually in equinus position as well (Figs. 7-A, 7-B, and 7-C).

The sex incidence was almost equal, with thirteen male and eleven female patients. The deformity was bilateral in one-third of the cases. In patients with

TABLE I

ASSOCIATED CONDITIONS IN THE LOWER LIMBS

Entity	Associated Condition	No. of Limbs
Tibia		
	Short	32
	Bowed	22
	Straight	10*
Femur (excluding 8 bilateral cases)	Incomplete absence	2
	Shortening	11
	No shortening	3
Foot		
	Deformed	20**
	Structurally complete	12**
	Valgus position	32

* Associated with complete roentgenographic absence of the fibula.
** Not related to the severity of the micromelia.

TABLE II

CLASSIFICATION OF ANOMALIES

Classification	No. of Patients: Coventry and Johnson	No. of Patients: Hospital for Sick Children
Type I: Unilateral (Fig. 7-A) Partial absence of the fibula Valgus, but no other deformities of the foot Slight bowing of the tibia Some shortening of the limb	3 (10%)	3 (12.5%)
Type II: Unilateral (Fig. 7-B) Complete or nearly complete absence of the fibula Valgus and other deformities of the foot Marked bowing of the tibia Severe shortening of the limb	10 (34.5%)	10 (41.7%)
Type III: Bilateral or else unilateral with distant anomalies (Fig. 7-C) (Included as Type III are Type I or II deformities of one or both lower extremities)	16 (55.2%)	11 (45.8%)
Totals	29	24

unilateral absence of the fibula, lesions were twice as common on the right. This greater incidence of right-side lesions was previously reported by Corner; it was noted also by us in patients with congenital absence of the radius [7].

Other congenital anomalies in addition to those observed in the lower extremities were noted in seven patients (Figs. 8-A through 8-C).

The patients were classified according to the types suggested by Coventry and Johnson (Table II). The incidence of the three types of anomalies in their series and in ours is remarkably similar. The criteria used for this classification by these authors are listed in Table II.

TREATMENT

It is almost axiomatic to say that there can be no standard treatment for

TABLE III

DATA ON TWENTY-FOUR PATIENTS

Classification	No. of Cases		Treatment	Average Follow-up (*Years*)	
Type I	3		Conservative; either no treatment, or else braces and manipulation	10	Satisfactory
Type II	10				
		6	Conservative; braces or manipulation or both	5	Satisfactory
		1	Partial fusion	2	Satisfactory when last seen
		1	Resected fibular anlage	1	No change
		1	Amputation, Syme's (patient seven years old)	15	Excellent
		1	Amputation, Syme's (patient seven years old)	5	Satisfactory
Type III	11				
		3	Soft-tissue operation and prosthesis	5	Satisfactory
		1	Resected fibular anlage	1	No change
		2	Tibial osteotomy	3	Rapid solid union
		1	Amputation, mid-calf	(lost)	
		1	Amputation, mid-calf	13	Ischial-bearing
		1	Amputation, mid-calf	6	Ischial-bearing; revision needed
		1	Amputation, modified Syme	2	Satisfactory
		1	Amputation, modified Syme	2	Satisfactory

this condition (Table III). The problem at hand is not simply an absent bone, but rather a dysplastic limb. Factors such as age, severity of the anomaly, and other distant abnormalities are obvious modifying considerations.

However, there are three common components—foot deformity, tibial bowing, and discrepancy in length of the limbs—which must be considered in treatment. All three of these may be present in the same patient, but usually one of them predominates and requires specific treatment.

Foot Deformity

Manipulations, applications of plaster casts, and tendon-lengthening procedures for marked foot deformities are followed by recurrence of the deformity unless the foot is overcorrected and maintained in that position. A posterolateral surgical release is necessary when the deformities are severe. The surgical procedure should then include partial excision of the fibrocartilaginous band and release of the foot below the fibular remnant (Fig. 9) by cutting of ligamentous and capsular structures that tend to maintain the foot in valgus position.

In the mild cases, the Type I deformities (Table II), the ankle joint is occasionally stable despite absence of the fibula (Fig. 7-A). In this event there is a good range of plantar and dorsal flexion at the talotibial joint, yet so little lateral motion that the foot does not displace into a more valgus position when weight is borne on this extremity. Under these circumstances, little or no treatment is necessary. However, stabilization of the ankle might be indicated on occasion, although this procedure has not been employed in this series. Needless to say, care should be taken not to disturb the lower tibial epiphysis when this is done, since shortening is always present to start with.

FIG. 5-A

FIG. 5-B

FIG. 5-C

Congenital bowing of the tibia. As with congenital absence of the fibula, the tibia is usually bowed anteriorly. However, the foot is never deformed even though it may be displaced. Roentgenograms will establish the diagnosis, since, with congenital bowing of the tibia, the fibula is present and the foot is normal.

Bowing of the Tibia

Tibial bowing, *per se*, rarely presents any difficulty. It accounts for little of the shortening and except for the medial bowing has little deleterious effect on the foot and ankle.

Thompson, Straub, and Arnold have drawn attention to the fibular anlage (Fig. 9). When present, it can be palpated subcutaneously, but it is rarely visualized by roentgenogram. They recommended early excision of this fibrous tissue to encourage subsequent correction of tibial bowing, theoretically, by freeing the tibia from this tethering band. Our follow-up of patients so treated does not as yet permit any conclusions concerning the value of this procedure, but it must be remembered that spontaneous correction of anterior tibial bowing will occasionally take place (Figs. 3-A and 3-B).

No spontaneous correction of medial bowing has been noted. On the other hand, progression of bowing does not occur. If the tibia is straight initially, bowing does not develop with growth. Braces and weight-bearing do not appear to influence the bowing.

A tibial osteotomy was performed for severe bowing on two occasions. The correction obtained at operation in these instances has been maintained during growth to date. In doing this procedure it is important to lengthen the soft tissues, including the fibular anlage and the tendo achillis, to allow full correction. Delayed tibial union after osteotomy need not be feared in these cases, in contrast to congenital bowing when in infancy osteotomy does result in pseudarthrosis.

FIG. 6-A

Congenital absence of the tibia. This deformity is much less common than congenital absence of the fibula. It presents a different clinical picture, with the foot in varus position and the knee dislocated. The fibula is not severely bowed, and the anomalies of the foot are usually located on the medial side.

FIG. 6-C

FIG. 7-A

FIG. 7-B

FIG. 7-C

Figs. 7-A through 7-C: Classification of cases of congenital absence of the fibula, according to that of Coventry and Johnson.

Fig. 7-A: Type I. The anomaly is unilateral and consists of partial absence of the fibula, valgus but no other deformities of the foot, slight bowing of the tibia, and some shortening of the limb. Note that the foot does not displace appreciably into valgus position even though the right leg is bearing weight.

Fig. 7-B: Type II. The anomaly is also unilateral, but is more severe with complete or nearly complete absence of the fibula, valgus and other deformities of the foot, marked bowing of the tibia, and severe shortening of the limb.

Fig. 7-C: Type III. The anomaly is either bilateral, with the same features as in Types I and II, or else it is unilateral and associated with distant anomalies.

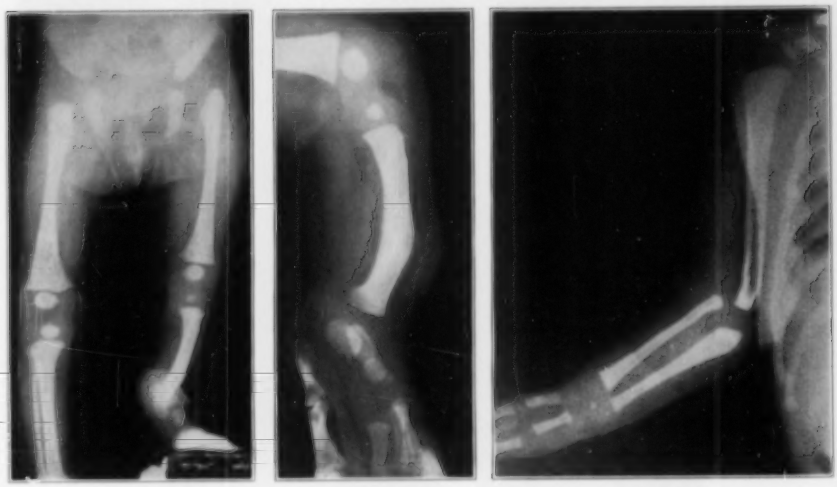


FIG. 8-A FIG. 8-B FIG. 8-C

Figs. 8-A through 8-C: Roentgenograms of Type III congenital absence of the fibula with associated congenital anomalies.

Figs. 8-A and 8-B: The lower left extremity shows: congenital dislocation of the hip; congenital absence of the fibula with tibial bowing; tibial shortening; and a deformed and displaced foot.

Fig. 8-C: The upper right extremity shows: adactylia; syndactylia; and congenital dislocation of the head of the radius.

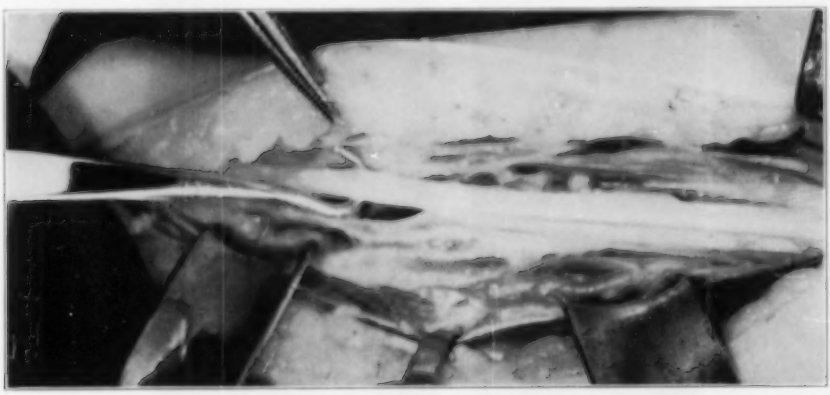


FIG. 9

Fibular remnant. The fibrocartilaginous band is continuous with the lower end of the fibula when the latter is present; the peroneal muscles originate from the band at its proximal end, and the interosseous membrane is attached to its medial aspect.

Discrepancy in Length of the Limbs

Discrepancy in the length of the limbs can be the most important and distressing aspect of this deformity. A limb with an absent fibula is always short.

FIG. 10-A

FIG. 10-B

Roentgenogram and photograph showing severe anomalies of both lower extremities (Type III). In this instance, early amputation is recommended. Good weight-bearing stumps can be fashioned on each side, and the shortening can be compensated for by the prosthesis. Note the displacement of the calcaneus and the severe deformity in both feet. The dimple over the apex of the tibial bow is well visualized.

FIG. 11-A

FIG. 11-B

Fig. 11-A: Any amputation should preserve as much length as possible; the lower tibial epiphysis should be saved when the tibia is straight.

Fig. 11-B: The calcaneus may be implanted over the distal end of the tibia and provide a good end-bearing stump.

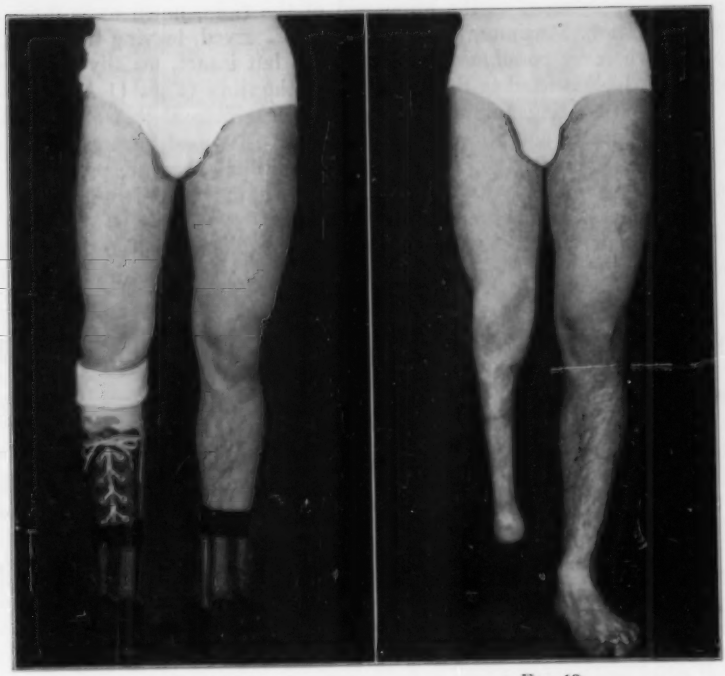


FIG. 12 FIG. 13

Fig. 12: Shortening is not a problem. The gait is almost normal, and the deformity is usually unnoticed.

Fig. 13: A Syme amputation was performed fourteen years previously, when the patient was seven years old. The foot was deformed, and the extremity was short.

The discrepancy may be mild, in which case it may be compensated for by an equinus foot or the use of a raised heel. Later, it can be corrected by an epiphyseal arrest on the opposite side. The shortening, however, may be grotesque when this deformity is associated with an absent femur. A prosthesis will then be required, and early foot amputation will simplify prosthetic fitting. When the length discrepancy is between these extremes, that is, three to five inches, the solution is more controversial. Epiphyseal arrest is ill-advised if there is no hope of satisfactory equalization; shortening a normal limb to conform to a misshapen limb will not appeal to parents.

Hence, early treatment has consisted of a raised surgical boot. Early amputation is often contemplated but is almost always deferred. It is suggested that amputation should be performed earlier and more frequently.

Early amputation is recommended whenever length discrepancy is expected to be more than three inches at the completion of growth. A second indication for early amputation is a severe foot deformity (Figs. 10-A and 10-B). In either instance the operation will provide esthetic and functional improvement, since length discrepancy will be corrected by the prosthesis (Fig. 12).

An infant will walk with a prosthesis quickly and well. His adaptation is automatic, and there is no evidence of emotional shock. Also, the cost and wearing of a prosthesis should not be serious objections. Any surgical shoe with a raise of three inches or more is, in fact, far uglier than the prosthetic appliances now available for the amputation stumps of these patients.

Seven amputations were performed because of severe shortening. The operation of choice was the Syme amputation, usually modified by preserving the

calcaneus and applying it to the lower end of the tibia, much as is done in a Pirogoff amputation. Maximum tibial length is preserved, leaving the lower tibial epiphysis wherever possible. The heel pad is left intact, usually attached to the calcaneus which is fixed to the lower end of the tibia (Figs. 11-A and 11-B). Unlike other end-bearing amputations in growing children, this stump, like a normal heel, is durable and allows growth (Fig. 13). If tibial bowing is severe, it should be corrected by shaving the crest of the tibial bow, or, preferably, by performing a wedge osteotomy. A below-the-knee amputation should not be performed simply because of severe tibial bowing.

A regular Syme prosthesis is well tolerated by older children, but a pelvic belt and a thigh harness are necessary for infants and young children.

CONCLUSION

A limb with an absent fibula is truly a limb dysplasia, with serious anomalies in the foot, the tibia, and the femur.

Tibial bowing is not affected by weight-bearing and braces. It may regress spontaneously, but will not progress.

Tibial osteotomies will unite.

The treatment required must be specific for each patient. It is suggested, however, that amputation should be performed early whenever shortening of more than three inches is anticipated.

Every effort should be made to perform a Syme type of amputation because the heel pad provides a durable stump which lengthens with growth.

REFERENCES

1. Bagg, H. J.: Disturbance in Mammalian Development Produced by Radium Emanation. Am. J. Anat., **30**: 133-161, 1922.
2. Bardeen, C. R., and Lewis, W. H.: Development of the Limbs, Body-Wall and Back in Man. Am. J. Anat., **1**: 1-36, 1901.
3. Borggreve, J.: Cited by Van Nes.
4. Corner, E. M.: The Clinical Picture of Congenital Absence of the Fibula. British J. Surg., **1**: 203-206, 1913.
5. Coventry, M. B., and Johnson, E. W., Jr.: Congenital Absence of the Fibula. J. Bone and Joint Surg., **34-A**: 941-955, Oct. 1952.
6. Duraiswami, P. K.: Experimental Causation of Congenital Skeletal Defects and Its Significance in Orthopaedic Surgery. J. Bone and Joint Surg., **34-B**: 646-698, Nov. 1952.
7. Farmer, A. W., and Laurin, C. A.: Congenital Absence of the Radius. Canadian J. Surg., **1**: 301-308, 1958.
8. Gegenbaur, Carl: Elements of Comparative Anatomy. Translated by E. J. Bell and Revised by E. R. Lankester, p. 473. London, Macmillan and Co., 1878.
9. Thompson, T. C.; Straub, L. R.; and Arnold, W. D.: Congenital Absence of the Fibula. J. Bone and Joint Surg., **39-A**: 1229-1236, Dec. 1957.
10. Van Nes, C. P.: Rotation-Plasty for Congenital Defects of the Femur. Making Use of the Ankle of the Shortened Limb to Control the Knee Joint of a Prosthesis. J. Bone and Joint Surg., **32-B**: 12-16, Feb. 1950.
11. Warkany, Joseph; Nelson, R. C.; and Schraffenberger, Elizabeth: Congenital Malformations Induced in Rats by Maternal Nutritional Deficiency. III. The Malformations of the Extremities. J. Bone and Joint Surg., **25**: 261-270, Apr. 1943.

DISCUSSION

Dr. T. Campbell Thompson, New York, N. Y.: I am quite convinced that early excision of the thick fibrous band in the posterolateral portion of the limb is worth while and will largely correct the equinus position of the foot and the bowing of the tibia. The patient can then get along very well with some type of appliance—a raised shoe, a special brace, or a prosthesis.

There is no question that in patients with a normal hip and thigh the best ultimate result is a Syme amputation because the shortening is almost always more than five inches. This amputation is far superior to an ordinary below-the-knee amputation, as the short below-the-knee stump can be fitted with a prosthesis that matches the normal limb and still allows full end-bearing. The only question is whether it should be done at an early age. Should one wait until the patient has attained full growth when a good Syme stump can be assured?

The advantages of early amputation have been presented by the authors. We all know that congenital amputations can be well fitted at an early date and there is no trouble with the stumps. However, below-the-knee stumps, after trauma in young children, are often

(Continued on page 22)

The Pathomechanics of the Knee Joint

I. The Medial Collateral Ligament and Lateral Tibial Plateau Fractures *

BY ALBERT F. MARTIN, M.D., SALT LAKE CITY, UTAH

From the Departments of Orthopaedic Surgery, University of California Medical School and the Samuel Merritt Hospital, Oakland, California

The role played by the medial collateral ligament of the knee in fractures of the lateral tibial plateau is not well understood. Authorities vary in opinion regarding the extent of ligamentous injury sustained around the knee in depressed fractures of the lateral plateau. The most prevalent concept seems to be that either the ligamentous structures yield medially or the bone is fractured, one to the exclusion of the other. Apparently, diagnosis of a torn ligament is rarely made in the presence of fracture, for I found but one recorded instance of a plateau fracture in which repair of a torn collateral ligament was accomplished [8].

The studies of Brantigan and Voshell [1,2] have materially helped in understanding the relative importance of the various ligaments in the knee mechanism, and the work of O'Donoghue emphasized the need for early repair of torn ligaments around the knee. Thus, with plateau fractures, any derangement in joint mechanics produced by the fracture would be compounded by damage to the medial collateral ligament. This is particularly true when the ligamentous injury results in valgus instability of the knee. In such cases it is probable that early diagnosis and repair of a torn ligament would improve the end result. Early diagnosis has been almost precluded in most cases by the difficulties encountered in detecting evidence of severe damage to the medial collateral ligament. Local signs, such as ecchymosis and tenderness over the medial collateral ligament and palpable opening of the medial joint space with abduction at the knee, are helpful, but the need for more exact methods of diagnosis is apparent.

The study herein presented is an attempt to establish a test by which the integrity of the medial collateral ligament can be determined in the presence of a depressed fracture of the lateral plateau. The test proposed consists in applying stress to the knee and recording the resultant deformity on roentgenograms. Twenty-two knees with injuries to the ligaments caused by athletics were subjected to this test and then surgically explored. The pathological condition encountered was correlated with the findings of the test. Structural tests were also made on freshly amputated limbs. From the analysis of these data, some theoretical considerations are presented concerning the mechanics of knee injuries.

METHODS AND OBSERVATIONS

The knee joints of twelve fresh amputation specimens were dissected and abduction experiments performed. In each case the collateral ligaments were exposed and the knees mounted to fix the femur rigidly in a wooden wedge mount. Thus, full extension was maintained and rotation prevented during the tests. Three sets of abduction stress roentgenograms were made of each specimen: first, with intact bone and ligamentous structures; second, after resection of the lateral tibial plateau and meniscus; and last, after section of the medial collateral ligament with or without section of the cruciate ligaments.

The lateral tibial plateau was resected transversely at approximately the level of the fibular head as far medially as the intercondyloid eminence. This was accomplished through a window in the anterolateral capsule, and a section of

* Read at the Annual Meeting of the Western Orthopedic Association, Portland, Oregon, October 25, 1958.

TABLE I

ABDUCTION STRESS IN THE FRONTAL PLANE

Knee No.	Medial Clear Space in Millimeters (Measured 5 Millimeters from Tibial Edge)			
	Normal Knee	Plateau Resected	Collateral Ligaments Cut	Difference
1	6.0	6.5	12.0	6.0
2	10.0	11.0	16.0	6.0
3	9.0	10.0	15.0	6.0
4	8.0	8.5	11.0	3.0
5	10.0	10.5	15.0	5.0
6	7.0	7.5		
7	9.0	9.5		
8	2.5	5.0	7.0 (C)*	4.5
9	5.5	6.0	7.5 (C)	2.0
10	6.0	7.0	15.0 (C)	9.0
11	6.0	6.0	8.0 (C)	2.0
12	6.0	7.0	10.0 (C)	4.0

*(C) indicates that the cruciate ligaments were also sectioned.

bone and articular cartilage of sufficient size was removed so that contact between the lateral femoral condyle and the surface of the tibial defect was not possible on abduction of the knee (Fig. 1). Care was taken to preserve the attachments of the cruciate ligaments. In the cases where the medial collateral ligament was sectioned, this was performed at the level of the superior edge of the medial meniscus and all of its components were transected: the straplike, deep portion, the superficial oblique fibers, and those portions of the capsule with which the ligament is fused. The capsule was otherwise undamaged on the medial side of the joint.

Figure 2 is a series of superimposed tracings of the roentgenograms of one specimen which shows the effect of each step in the experimental procedure. Note

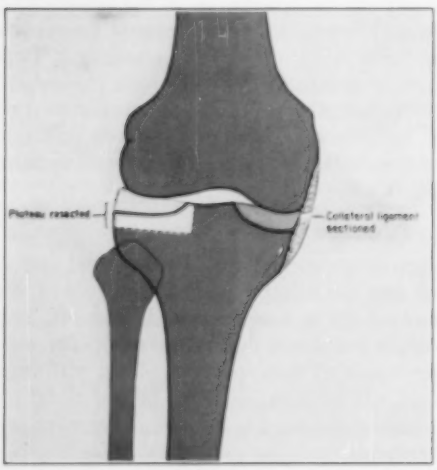


FIG. 1

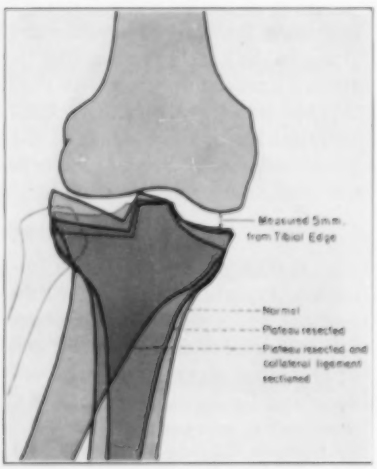


FIG. 2

Fig. 1: Drawing showing the site of tibial resection, removal of meniscus, and sectioning of the medial collateral ligament.

Fig. 2: Superimposed tracings of roentgenograms made at each step in the experimental procedure. The medial clear space of the joint was measured at a point five millimeters lateral to the tibial edge.

that regardless of plateau resection, there is little opening of the medial clear space until the medial collateral ligament is sectioned. The medial clear space is defined as the radiolucent area between the medial femoral condyle and the subjacent tibia. It was measured in each case at a point five millimeters lateral to the edge of the tibial plateau.

The clear space was measured on the roentgenograms of the abducted specimens (Table I). The knee joints were abducted manually with sufficient force to produce a state of mild tension on the medial collateral ligament. Gentle manual abduction was considered necessary since greater stress than this might, in the clinical application of this test, do further damage to a fractured lateral tibial plateau. Changes in the medial clear space were believed to be more accurate indicators than changes in valgus position at the knee because the errors caused by knee flexion, rotation of the limb, or obliquity of the x-ray beam, are greatly minimized when measuring changes in the clear space. (The results are presented with each step tabulated in columns.) With resection of the plateau alone there was only one instance (Specimen 8) in which the medial clear space opened more than 1.0 millimeter. In all specimens after section of the medial collateral ligament, the clear space increased markedly. Careful inspection of Specimen 8 failed to reveal why the medial clear space opened 2.5 millimeters after merely resecting the lateral plateau.

Observations were also made concerning the role of the cruciate ligaments in preventing abduction under these experimental conditions. Brantigan and Voshell showed that in the normal knee the cruciate ligaments will prevent abduction when the knee is fully extended. Under normal conditions the lateral plateau serves as both a fulcrum and a lever arm by which tension is applied to the cruciate ligaments. After resecting the lateral tibial plateau the fulcrum is removed and the cruciate ligaments serve more as a suspensory mechanism when the knee is abducted. Even though the lateral tibial plateau is resected with resulting loss of stability in the knee joint, the cruciate ligaments continue to serve as a flexible hinge holding the tibia to the femur and minimizing lateral subluxation of the tibia. When abduction stress is applied to the knee after resection of the lateral tibial plateau, the cruciate ligaments tighten and permit more effective leverage at the medial joint space. This places the abduction fulcrum or axis of rotation during abduction at the lateral aspect of the medial femoral condyle. Evidence to support this observation is shown in Table I. In five of the twelve knees the cruciate and medial collateral ligaments were sectioned simultaneously. In these knees there was usually less opening of the medial clear space than in the joints in which the cruciate ligaments were intact. The best explanation for this is that the tibia, having lost its suspension to the femur, is subluxated or displaced laterally rather than rotated into abduction. Knee 10 was exceptional since the observed opening of its medial clear space was the largest recorded, even though the cruciate ligaments were sectioned. The reason for this is not clear. Attempts to demonstrate roentgenographically the actual amounts of subluxation of the tibia on the femur after division of the cruciate ligaments were unsuccessful because of the small distances involved and the absence of reliable landmarks.

To correlate the experimental data with clinical findings, twenty-two knee injuries were studied in which there was rupture of both the medial collateral and the anterior cruciate ligaments, as proved at operation by Hitchcock and his associates at the Cowell Memorial Hospital, Berkeley, California. In each instance general or spinal anesthesia was utilized as an aid in obtaining preoperative abduction stress roentgenograms of both the uninjured and the involved knee. The medial clear spaces were measured as in the experimental studies and the values recorded. The mean difference between the heights of the normal and abnormal knees was $5.3 \pm .92$ millimeters *, a value quite comparable to that found in the experimental series. This indicates that opening of the medial clear space

* $P = .05$ after suitable statistical analysis.

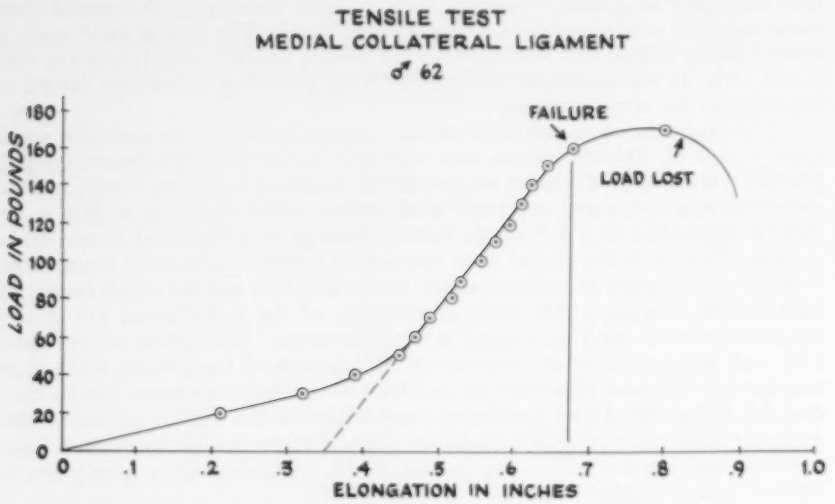


CHART I-A

occurs in knees having a torn medial collateral ligament regardless of the condition of the lateral plateau.

To obtain further information about the relative strengths of the medial collateral ligament and the lateral tibial plateau, two fresh knee specimens were

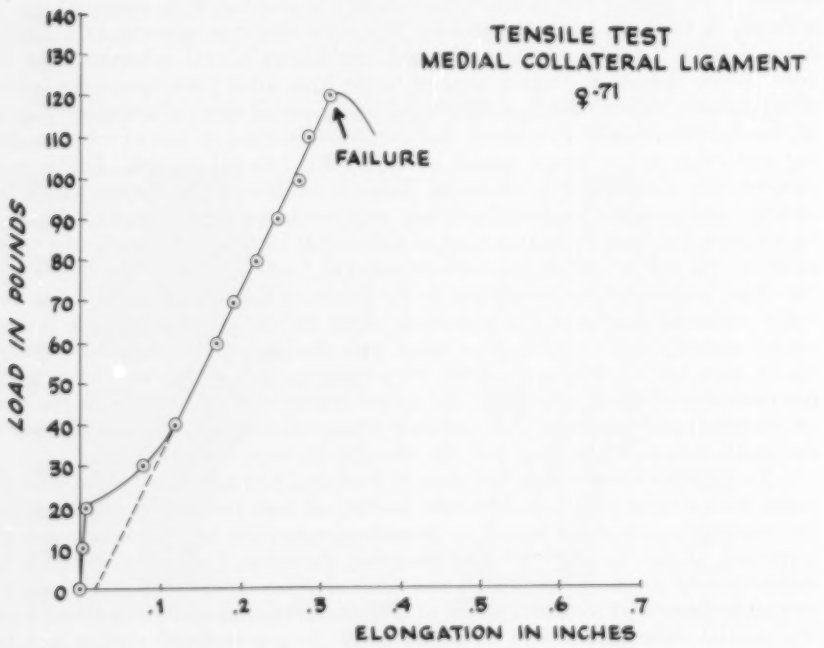


CHART I-B

Charts I-A and I-B: Load-deformation curves obtained in tensile testing of the two medial collateral ligaments. The triangular area beneath the straight line portion of the curve in Chart I-A was measured to determine energy consumption. A similar area was measured for each load-deformation curve.

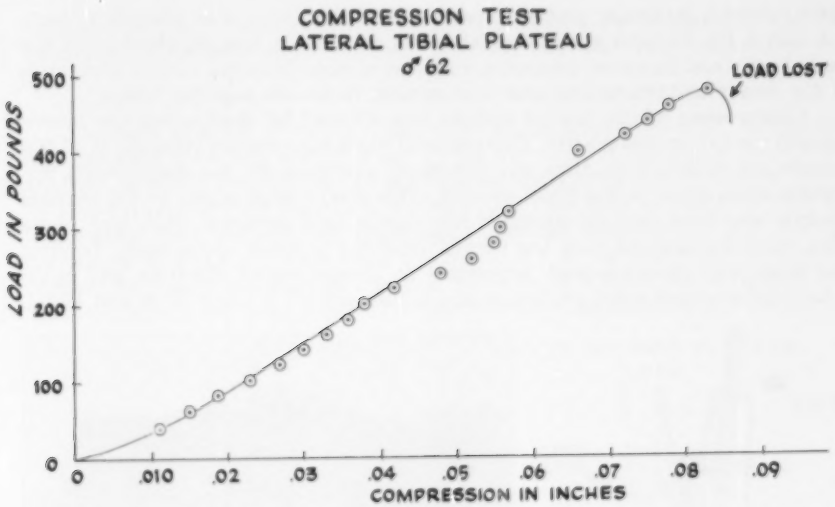


CHART II-A

subjected to tensile testing of the medial collateral ligaments and compression testing of the lateral tibial plateaus. These static tests provided individual evaluation of the two structures.

The tensile tests of the ligaments were performed on a universal testing machine by stripping the knees of all soft-tissue attachments except the medial collateral ligaments. Cold-rolled steel rods were then inserted into the femoral and tibial medullary canals and firmly transfixed with stove bolts across both cortices (Fig. 3). These specimens were loaded at ten-pound increments with

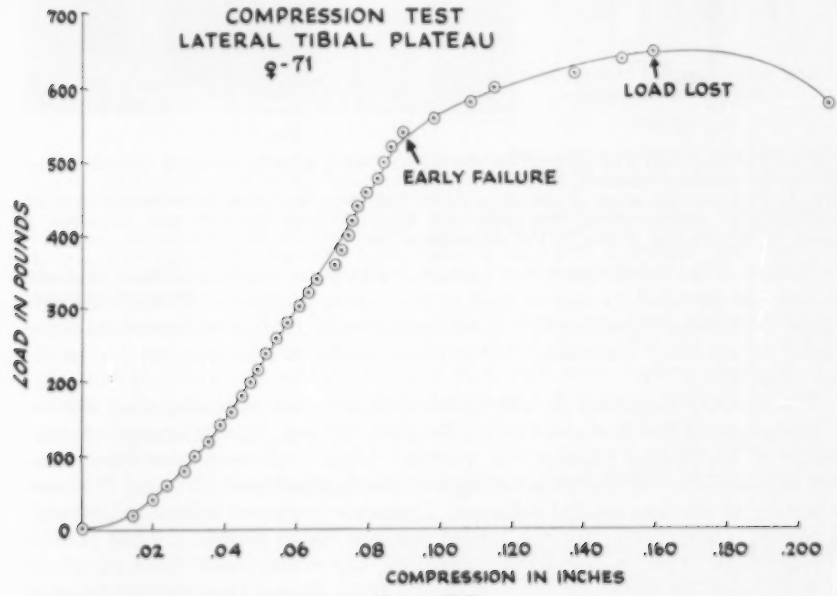


CHART II-B

Charts II-A and II-B: Load-deformation curves obtained in compression testing of two lateral tibial plateaus.

twenty-second intervals, and the load-deformation curve was plotted (Charts I-A and I-B). In both specimens failure in the tensile test occurred when the medial collateral ligament avulsed a fragment of bone from the medial epicondyle of the femur and started to tear transversely across its superior fibers.

Compression of the lateral plateau was effected by duplicating the lateral femoral condyle in tico plastic. This material has a compression strength of 15,000 pounds per square inch, thus any deformity produced by the compression apparatus must occur in the tibial plateau. This plastic duplication of the femoral condyle was then used to compress the rigidly held proximal tibia and fibula after carefully aligning it in the normal extended position of the knee. Loading was done with twenty-pound increments at twenty-second intervals and again a load-deformation curve plotted to the failure point * (Charts II-A and II-B).

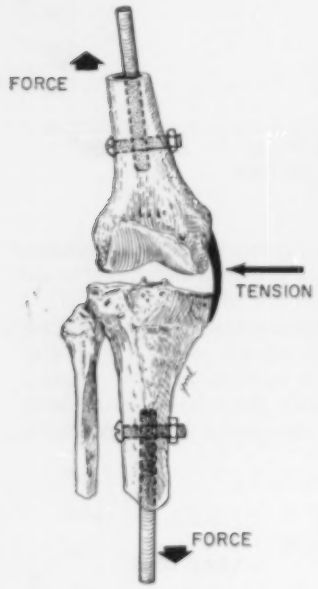


FIG. 3

FIG. 4

Fig. 3: The steel rods were grasped by the tensile-testing machine to apply tension to the intact medial collateral ligament.

Fig. 4: This roentgenogram of one knee shows that there was remarkable derangement of the subarticular weight-bearing bone trabeculae, although there was little gross evidence of damage at the time of failure of the compression test.

The failure of the compression test occurred with little visible evidence of damage, and was detected by loss of load on the testing apparatus. Roentgenograms of these compressed specimens revealed considerable damage to the subarticular weight-bearing bone trabeculae, which explains the loss in compression resistance (Fig. 4).

The methods employed do not represent pure materials testing since neither the dimensions of the medial collateral ligament nor the size of the area of compression of the lateral plateau are recorded. These test results are, therefore, solely representative of the total strength of the ligament and the bone. The tensile testing of the two medial collateral ligaments produced failure at 160 and 120 pounds, respectively, and the lateral plateaus failed at 480 and 540 pounds of compression. Thus, under static conditions, the compression force sustained was in one case 3.0 times and in the other 4.5 times greater than the tensile force

* In both the tensile and compression experiments the deformation was recorded by utilizing a dial gauge accurate to .001 inch. The accuracy of the testing apparatus is 3 per cent for the loads utilized,

sustained medially. Total elongation of the ligaments achieved was .329 and .290 inch, and compression of the plateaus was .083 and .090 inch, respectively †.

ANALYSIS OF FORCES IN THE FRONTAL PLANE

Dynamic analysis of energy dissipation at the knee joint with its supporting ligaments, bone, and muscle is far beyond the scope of this paper. Even static analysis, in which the problems of acceleration and inertia are disregarded, must be founded on carefully stipulated assumptions. This need not detract from the information thus derived, but conclusions must be tempered by the inherent limitations of static analyses when applied to the living body. By using static engineering principles on beam analysis, some small insight can be gained into the mechanics of the knee, especially regarding force distribution and the magnitude of these forces. The present analysis is based on these assumptions: (1) that the lower extremity is a rigid body, fixed and immovable at the hip and foot; (2) that the only supporting structure that can resist tension medially is the medial collateral ligament; (3) that the broad articulating surface between the lateral femoral condyle and the subjacent tibia may be represented as occurring at a single point of contact; and (4) that the shearing force at the knee is resisted by bone contact between the femur and tibia and by tension in the medial collateral ligament.

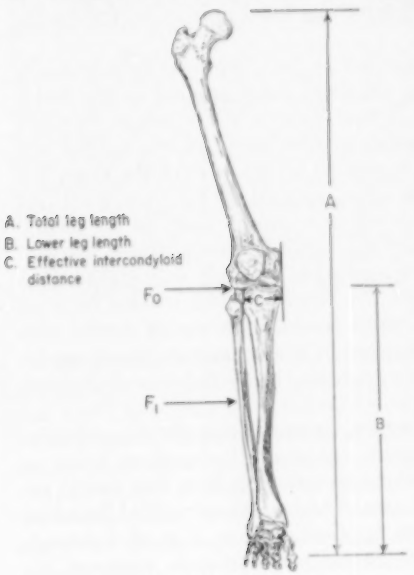


FIG. 5

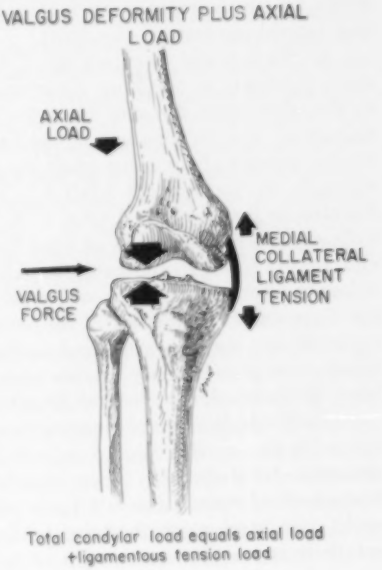

VALGUS DEFORMITY PLUS AXIAL LOAD
AXIAL LOAD
MEDIAL COLLATERAL LIGAMENT TENSION
VALGUS FORCE
Total condylar load equals axial load +ligamentous tension load

FIG. 6

Fig. 5: The factors governing load transmission to the medial collateral ligament and the lateral tibial plateau are based on lever-arm considerations. F_0 and F_1 refer to two possible sites for force application to the lateral aspect of the leg.

Fig. 6: If certain simplifying assumptions are made it can be said that a pure valgus force produces the same tension on the medial collateral ligament as it does compression of the lateral tibial plateau. On the other hand, when an axial load is added to the valgus force, the total load on the lateral condyle then equals the axial load and the load caused by the ligamentous tension.

If force is applied to the lateral aspect of the lower extremity, directed medially, in the frontal plane, and is perpendicular to the long axis of the limb (thus tending to produce a valgus deformity in the frontal plane) it will produce the

† The total ligamentous elongation was measured by extrapolating the straight line portion of the tensile curves back to the base line and recording the length from this point to that at the time of failure. This maneuver is necessary to eliminate the deformation produced by the gripping effect of the tensile testing machine at the early small loads.

same tension force on the medial collateral ligament as it does compression force on the lateral tibial plateau. If force is applied as just described, there remain four factors which govern the total load transmitted to the medial collateral ligament and the lateral tibial condyle. These are the total leg length, the length of the leg from the knee to the sole of the foot, the site of the applied force, and the distance from the point of contact of the lateral condyle of the femur with the tibia to the medial collateral ligament (Fig. 5). The specific load in terms of pounds per square centimeter (stress) cannot be calculated since the size of the area of contact between the femur and tibia and the cross-sectional area of the medial collateral ligament are unknown. However, the tolerable limits of applied force which the tibial plateau and the medial collateral ligament can sustain before failure have been determined for the experimental knees, and if the dimensions of the limb and the amount and site of the applied force were known, it would be simple ratio mathematics to determine the force applied to the medial and lateral aspects of the joint.

If the hip and knee are fixed, force applied at the knee level is potentially more injurious to the knee structures than an equal force applied to the femur or the tibia some distance from the knee. This is because the leverage (bending moments) will be greater and will thus inflict greater loads on the medial and lateral aspects of the joint when the force is applied at the level of the knee. For example, in the average adult leg, a given force, applied at the knee level and directed as just described (Fig. 5, F_1) will result in a tension force on the medial collateral ligament that is at least four times the applied force provided the foot and hip are fixed. In contrast, the same force applied to the tibia only eight inches below the knee (Fig. 5, F_2) will result in just two times as much tension force on the ligament (a reduction of 50 per cent). Such calculations are of course only approximate, since they depend on the dimensions of the leg and are based on the assumption that the only tension-resisting structure on the medial side of the knee joint is the medial collateral ligament.

These mathematical observations are helpful in correlating the site of damage with the mechanism of injury but, again, interpretative caution must be observed. By definition, force does not produce structural failure but rather reduces applied stress, which is force per unit area. Thus, a hypertrophied ligament could withstand a greater total tension-force application than a small ligament, yet their stress tolerance could be identical. Stress measurements were not attempted on the experimental knees since the major concern is the behavior of the tibia and medial collateral ligament as organs, not as tissues. The dimensions of the ligaments seem less important than determination of the force required to produce failure of the ligaments as a whole, regardless of size.

If at the time of valgus strain there is an increase in the axial load on the femur, plateau fracture would be more likely to occur than rupture of the medial collateral ligament because extra load will be borne by the lateral femoral condyle and the lateral tibial plateau. Under these circumstances the total load on the lateral condyle equals the axial load and the ligamentous tension load (Fig. 6). These conditions would obtain in a fall from a height with the leg abducted. Conversely, under circumstances where there is no axial load at the time of valgus strain, fracture of the lateral plateau would be much less likely to occur than simple rupture of the medial collateral ligament. This has been shown experimentally by Hönigschmied, who forcibly abducted thirty-one knee specimens without axial load; in twenty-eight, injury to the medial collateral ligament was produced first, and, then as the abduction continued, the cruciate ligaments and the capsule were torn. In only three of these specimens did fractures occur—two of the lateral plateau and one of the lateral femoral condyle. These experiments were performed on fixed cadaver specimens, the tissues of which were stiffened and of increased tensile strength, thus accounting presumably for the three fractures which did occur in pure abduction. Horwitz recorded a similar experience

with four knee specimens from human cadavera in which the medial collateral ligament tore in all cases without bone injury. Clinically, this is illustrated by the greater frequency of tears of the medial collateral ligament in situations where force is applied directly to the lateral aspect of the knee with the patient on the ground. This is seen in some contact sports and in certain situations where there is virtually no axial load present.

Concerning the mechanism of fractures of the lateral plateau, a few observations derived from the static loading experiments seem pertinent. It is essential to recall, however, that most injuries occur under dynamic rather than static conditions, and conclusions based on these experiments must be made with reservations. With static loading force is exerted, which is expressed as pounds, whereas with dynamic loading energy is generated, which is expressed as foot-pounds. The two are not always directly comparable as Evans has so vividly shown. However, in dealing with bone tissues, Evans and associates have also shown that data obtained from static tests are comparable to those derived from dynamic experiments. Accordingly, the load-deformation curves obtained in these static tests were analyzed from the standpoint of energy consumed by the ligaments and the lateral plateaus. These values were derived by calculating the areas under the curves (Charts I-A through II-B) which mathematically represent energy. Thus, the ligaments consumed 52.8 and 34.8 pound-inches of energy, respectively, compared with 43.2 and 37.8 pound-inches for the lateral plateaus. This shows that the energy-absorbing capacity of the medial collateral ligaments and the tibia was quite comparable on the specimens tested. This helps to explain why concomitant injury of both structures can occur.

DISCUSSION

Observations from the experimental stress roentgenograms supported two significant conclusions: First, increase in the medial clear space after plateau resection alone is minimum, there being only one instance where it was greater than 1.0 millimeter. This observation is of fundamental importance because it demonstrates that significant opening of the medial clear space will not occur with plateau injury alone. Second, and as a corollary, where there is an increase in the medial clear space of greater magnitude than 1.0 millimeter, it is highly suggestive of damage to the medial collateral ligament. It is essential that any increase in the medial clear space of an involved knee be compared with that of the normal knee, which is stress tested in a comparable position. Testing of the normal knee provides a control. This is necessary because of the individual normal variation in laxity of the medial collateral ligament. Thus, in cases of bilateral plateau fracture of the tibia this test is invalid.

Hohl and Luck found poor correlation between the depth of plateau depression and valgus deformity. They also demonstrated that valgus instability of the knee persisted in several cases of completely undisplaced lateral plateau fractures. On the basis of the data presented here, this implies that in these cases a concomitant tear or stretching of the medial collateral ligament must have occurred with the plateau fracture.

It is reasonable to conclude that patients with the combined injuries of fracture of the lateral tibial plateau and tearing of the medial collateral ligament may obtain a better clinical result if the tear is repaired early. Such a tear can be safely demonstrated prior to reduction by the abduction stress test. General or spinal anesthesia is necessary, because local anesthesia in the area of tenderness does not allow sufficient muscle relaxation for an adequate test. Abduction manipulation, if done gently, will not damage the lateral plateau and will not jeopardize reduction of the fracture.

SUMMARY

On the basis of the experimental data presented, it seems logical to conclude that fracture of the lateral tibial plateau and rupture of the medial collateral

ligament may occur simultaneously. In such circumstances, the medial collateral ligament injury may be demonstrated by gentle abduction stress testing of both knees and comparing the medial clear space of both the normal and the involved knees. Using this test, an increase of more than 1.0 millimeter in the medial clear space is presumptive evidence of either partial or complete rupture of the medial collateral ligament.

REFERENCES

1. Brantigan, O. C., and Voshell, A. F.: The Mechanics of the Ligaments and Menisci of the Knee Joint. J. Bone and Joint Surg., **23**: 44-66, Jan. 1941.
2. Brantigan, O. C., and Voshell, A. F.: The Tibial Collateral Ligament: Its Function, Its Bursae, and Its Relation to the Medial Meniscus. J. Bone and Joint Surg., **25**: 121-131, Jan. 1943.
3. Evans, F. G.: Stress and Strain in Bones: Their Relation to Fractures and Osteogenesis. Springfield, Illinois, Charles C. Thomas, 1957.
4. Evans, F. G.; Pedersen, H. E.; and Lissner, H. R.: The Role of Tensile Stress in the Mechanism of Femoral Fractures. J. Bone and Joint Surg., **33-A**: 485-501, Apr. 1951.
5. Hohl, Mason, and Luck, J. V.: Fractures of the Tibial Condyle. A Clinical and Experimental Study. J. Bone and Joint Surg., **38-A**: 1001-1018, Oct. 1956.
6. Hönigschmied, Johann: Leichenexperimente über die Zerreissungen der Bänder im Kniegelenk. Deutsche Zeitschr. f. Chir., **36**: 587-620, 1893.
7. Horwitz, M. T.: Injuries of the Ligaments of the Knee Joint. An Experimental Study. Arch. Surg., **38**: 946-954, 1939.
8. Knight, R. A.: Treatment of Fractures of the Tibial Condyles. Southern Med. J., **38**: 246-254, 1945.
9. O'Donoghue, D. H.: An Analysis of End Results of Surgical Treatment of Major Injuries to the Ligaments of the Knee. J. Bone and Joint Surg., **37-A**: 1-13, Jan. 1955.

DISCUSSION

Congenital Absence of the Fibula

(Continued from page 12)

unsatisfactory and give trouble for many years because the small bones grow through the skin of the sharply tapered stump end. The troubles often continue, and an above-the-knee amputation is frequently the end result.

I have only seen two patients with congenital absence of the fibula who have had a Syme amputation at an early age. Both these patients were having difficulty and will continue to have difficulty similar to that experienced by all amputees with poor below-the-knee stumps.

I hope the follow-up of the patients reported today by Dr. Farmer and Dr. Laurin will continue long after they have ceased to be children, because if a Syme amputation can be done in early childhood which will also give a good and painless stump that will last for the patient's lifetime, then the preferred treatment of these patients will be that outlined by the authors.

The treatment of patients with partial or complete absence of the femur (besides the absent fibula) is more difficult. In some instances one might even consider a turn-up-plasty amputation in order to obtain a good thigh stump.

Acute Suppurative Arthritis of the Hip in the Neonatal Period*

BY BENJAMIN E. OBLETZ, M.D., BUFFALO, NEW YORK

From the Children's Hospital, Buffalo

Suppurative arthritis of the hip joint is always a serious disease, but it is especially so in newborn infants during the first few weeks of life. The disease is difficult to diagnose in this neonatal period, and any delay in diagnosis and treatment may result in irreparable destruction of the hip joint.

From 1946 to 1956, fifteen infants in the neonatal period were treated for suppurative arthritis of the hip at the Children's Hospital in Buffalo. Of these, nine were full-term and six were premature infants with birth weights of from two pounds, eleven ounces, to four pounds, ten ounces. This report is based on a study of these fifteen infants.

Suppuration of the hip joint in these infants is caused by blood-borne organisms. Although septicemia is not uncommon in this age group, it is difficult to recognize. The classic signs of fever, chills, sweats, and prostration are not seen in these infants. Pediatricians at our hospital, after reviewing approximately 100 cases of septicemia in 2,000 premature infants, have come to suspect septicemia on the basis of the following clinical observations: presence of an open wound or known focus of infection; general observation that the infant is doing poorly; refusal or regurgitation of feeding; cyanosis during feeding; lethargy or irritability; increasing icterus; abdominal distention; appearance of edema in the lower extremities, buttocks, or genitalia; failure to maintain weight or to continue to gain when the infant had already been gaining; rapid fall in hemoglobin; and rise in temperature.

If septicemia is suspected, blood cultures should be made and appropriate antibiotics started at once. When the organism is identified and antibiotic sensitivity is determined, the most effective antibiotic should be used. Supportive treatment includes adequate caloric and fluid intake and blood transfusions if the hemoglobin has dropped below ten grams per 100 milliliters. In the presence of septicemia, skeletal localization of infection should be suspected and frequent careful examinations of the extremities should be made.

Infection of the hip joint may appear almost simultaneously with the onset of septicemia or later, even though the infant is on antibiotic therapy. The hip joint may become involved by hematogenous implantation of the synovial membrane or by direct extension from an osteomyelitic focus, either in the intracapsular portion of the femur or in the pelvic bones that form the acetabulum. Although the head and neck of the femur in the newborn infant are cartilaginous, a small strip of the bone metaphysis lies within the capsule. Osteomyelitis originating in this metaphyseal strip can break through the cortex and liberate pus into the hip joint (Fig. 1).

Involvement of the hip joint must be suspected in an infant with clinical manifestations of septicemia, as previously stated, and in addition one or more of the following: pain reaction, elicited by palpation of the hip or by passive movement of the extremity; unilateral edema or swelling of an extremity, buttock or genitalia; lack of active movement of the lower extremity; asymmetrical thigh and buttock creases; and abnormal position of the femur in flexion and adduction, with a bulge over the buttock.

* Read at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 26, 1957.

TABLE I

SIX PREMATURE INFANTS—CLINICAL CONDITION

Patient	Birth Weight	Age at Onset of Septicemia (*Days*)	Focus of Infection	Antibotics Given for Focus of Infection
D.B.	2 lbs., 11 oz.	24	Diarrhea	Yes
M.S.	2 lbs., 14 oz.	12	Scalp wound	Yes
P.K.B.	2 lbs., 13 oz.	42	?	No
M.H.	4 lbs., 7 oz.	27	Eye (?)	No
K.K.	4 lbs., 10 oz.	16	Umbilicus	No
D.C.	3 lbs., 6 oz.	30	Skin pustules	No

TABLE II

SIX PREMATURE INFANTS: TIME OF TREATMENT AND RESULT

	Time (Days) Elapsed					
Patient	From Clinical Onset of Septicemia and Treatment of Septicemia	From Clinical Onset of Septicemia to Onset of Hip Infection	From Onset of Hip Infection	From Onset of Hip Infection to Surgical Drainage of Hip	Delay in Treatment by Surgery or Aspiration	Fate of Head of Femur
D.B.	5	6	2	4	4	Disappeared
M.S.	7	14	6	6	6	Disappeared
P.K.B.	5	5	6	7	7	Disappeared
M.H.	9	8	8		8	Disappeared
K.K.	1	2	3		3	Coxa vara
D.C.	5	5	4		4	Died

As soon as suppuration is suspected, aspiration of the hip must be performed to establish the diagnosis and to identify the organism so that the most effective antibiotic can be used in treatment. If no pus is found, the aspiration should be repeated in a few hours. If pus is aspirated, penicillin should be instilled into the joint, especially if there is to be any delay in evacuating pus by surgical drainage. Prompt surgical drainage is imperative to prevent destruction of the femoral head.

TABLE III

NINE FULL-TERM INFANTS

Patient	Age at Onset of Hip Infection (*Days*)	Focus of Infection	Antibiotics for Focus of Infection	Fever at Onset of Hip Infection	Organism in Blood Stream	Organism in Hip Joint
E.D.	16	Umbilicus	No	?	Staph. a.	Staph. a.
R.F.	16	Abscess on foot	Yes	?	None	Staph. a.
D.P.	11	Umbilicus	Yes	Yes	None	Staph. a. and Strep. h.
S.R.	4	?	No	?	Staph. a.	Staph. a.
D.W.	8	?	No	?	Staph. a.	Staph. a.
D.H.	12	Diarrhea	Yes	?	Staph. a.	B. coli
R.M.*	28	Wound infection	Yes	101 degrees	No culture made	Staph. a.
J.Z.	28	?	Yes	?	Staph. a.	Staph. a.
N.R.	10	Umbilicus	No	99 degrees	Strep. h.	Strep. h.

*Bilateral involvement.

TABLE I—*Continued*

SIX PREMATURE INFANTS—CLINICAL CONDITION

Fever At Onset of Septicemia	At Onset of Hip Infection	Weight Loss during Septicemia	Organism Recovered	Bone Involved
None	101 degrees	Yes	Staph. a.	Femur
None	None	No	Staph. a.	Ilium
100 degrees	None	Yes	B. pyocyan.	Femur
100.6 degrees	100.2 degrees	Yes	Staph. a.	Femur
None	101.6 degrees	Yes	Staph. a.	Femur
None	None	No	Staph. a.	Femur

Roentgenograms of the hips are of great value in establishing the diagnosis, and, later, in following the course of the disease. In several instances, roentgenographic findings pointed to the diagnosis before septicemia or hip-joint infection was suspected on clinical grounds. These early findings included soft-tissue swelling and capsular distention (thirteen hips) and some degree of lateral and upward displacement of the femur.

In the absence of an ossified femoral head at this age, it is not possible to determine the location or integrity of the head by roentgenograms. Some degree of lateral and upward dislocation of the femur on the pelvis, indicating subluxation or dislocation, was seen on the roentgenograms of every one of the sixteen hips in the group of infants in this study (Fig. 2-A).

A focus of bone destruction indicating osteomyelitis may be seen early in this disease. This early bone involvement was noted in four patients; roentgenograms made on the day that suppuration was suspected indicated that the primary focus of bone infection preceded the liberation of pus into the joint.

Roentgenograms made later in the disease showed more definite evidence of osteomyelitis, first in the destructive phase, and later in the reparative stage. In eleven hips, the osteomyelitis was located in the femoral metaphysis (Fig. 2-B); in four hips, in the iliac portion of the acetabulum. There was no evidence of spread of the infection from the femur to the acetabulum or *vice versa*. In the one hip infected with *Streptococcus hemolyticus*, repeated roentgenograms showed no bone changes. This case may represent suppuration due to synovial

TABLE III—*Continued*

NINE FULL-TERM INFANTS

Time (Days) Elapsed					
From Onset of Hip Infection to Diagnosis	From Onset of Hip Infection to Surgical Drainage	From Onset of Hip Infection to Aspiration and Local Penicillin	Delay in Local Treatment (*Days*)	Bone Involved	Fate of Head of Femur
5	8		8	Ilium	Disappeared
7	16		16	Femur	Intact
6	8		8	Ilium	Deformed and subluxated
?	?		?	Femur	Coxa valga
16	16		16	Ilium	Disappeared
5		5	5	Femur	Coxa magna
4		4	4	Both femora	Disappeared
7		7	7	Femur	?
1		1	1	None	Intact

FIG. 1

Anterior photograph of the hip joint of a full-term stillborn infant, with the capsule opened and retracted to show the cartilaginous femoral head and the intracapsular portion of the bone metaphysis.

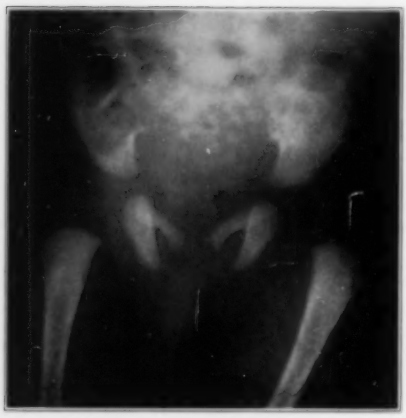


FIG. 2-A

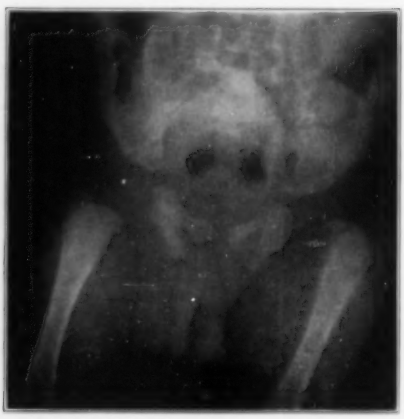


FIG. 2-B

Fig. 2-A: D. B. Roentgenogram of premature infant two days after onset of hip-joint infection, showing lateral subluxation of the left hip with soft-tissue swelling.

Fig. 2-B: Eleven days later. There is definite evidence of osteomyelitis of the femoral-neck metaphysis with periosteal stripping of the shaft and upward displacement of the femur.

implantation of organisms from the blood stream.

Later roentgenograms showed the final relationship of the femur to the pelvis, the fate of the femoral head, and the effects of the infection on the upper end of the femur (Figs. 2-C, 2-D, 3-A, and 3-B).

FIG. 2-C

One month after onset of infection. There is destruction of the neck of the femur, periosteal-bone formation, and upward displacement of the femur. Clinically, the infection had subsided.

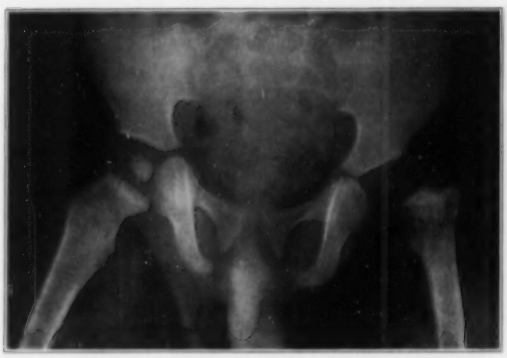


FIG. 2-D

One year after birth. The femoral head never appeared, having been destroyed by the infection. The femur is subluxated laterally and upward. Good bone texture has been restored to the upper shaft of the femur.

TREATMENT

If pus is found on aspiration, the hip joint must be drained to prevent destruction of the femoral head by the proteolytic enzymes liberated in the pus. According to Phemister: "If articular cartilage, immersed in pus produced by any of the pyogenic organisms, is incubated at a temperature of 55° C. so that bacterial action is suspended and proteolytic action augmented, the cartilage will be completely digested in a few hours." This necrotizing action may cause disintegration and disappearance of the femoral head with startling rapidity. Delay of even a few days may result in irreparable damage to the cartilaginous head and neck of the femur. Of nine hip joints infected with hemolytic *Staphylococcus aureus*, in which the suppuration went untreated for more than four days, seven femoral heads disappeared regardless of treatment by surgical drainage [4] or by aspiration and instillation of penicillin [3]. This emphasizes the need for early diagnosis. The penalty for delay in diagnosis or treatment is almost certainly a destroyed hip and a crippled child.

Some form of immobilization of the hip in the reduced position must be maintained until the infection is controlled and the femoral head can be demonstrated clinically, or, preferably, by roentgenogram, to be within the acetabulum. This may require gentle manipulation into abduction and immobilization in a double hip spica cast, usually in the frog-leg position. On occasion, the hips were immobilized in toe-toe-to-groin casts with an abduction bar; the period of immobilization varied from three weeks to eighteen months.

A long period of protection by immobilization is needed because retarded ossification may delay the roentgenographic appearance of the femoral head for many months. When it finally becomes evident that the femoral head has disappeared, immobilization is discontinued. The child may become ambulatory on the defective hip until a decision is made regarding the type of reconstructive hip surgery best suited to the individual problem.

Systemic treatment with antibiotics should be instituted as soon as the diagnosis of septicemia or hip-joint infection is suspected and continued for a

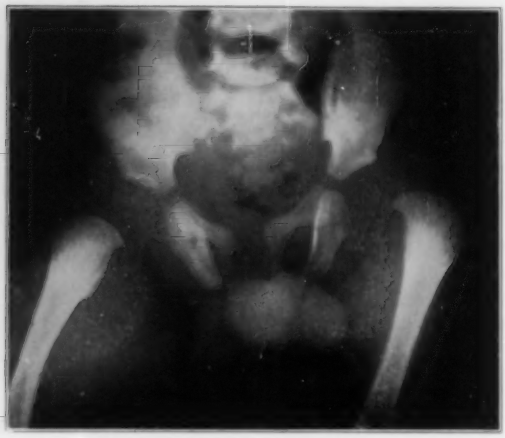


FIG. 3-A

R.M., a full-term infant, twenty-eight days old. This roentgenogram, made four days after onset of infection in both hip joints, shows lateral and upward displacement of both femora. (Later roentgenograms showed osteomyelitic changes in the metaphysis of each femur, similar to Figs. 2-B and 2-C.)

FIG. 3-B

Six years after both femoral heads had been destroyed by the acute infection in the neonatal period.

few weeks after all clinical signs of infection have subsided. Penicillin in large doses of two to 8,000,000 units daily, preferably by constant intravenous drip, will be well tolerated by even a small, premature infant. Streptomycin is given in doses of ten milligrams per kilogram of body weight every eight hours. When the organism is identified and antibiotic sensitivity studies are completed in the laboratory, this penicillin and streptomycin program can be changed if necessary. (If a penicillin-resistant staphylococcus is identified our bacteriologist, Dr. Erwin Neter, recommends treatment with vancomycin, kanamycin, or ristocetin.)

There were no instances of chronic osteomyelitis with draining sinuses or sequestrum formation. In two infants there were metastatic skeletal lesions outside the hip; one in the opposite tibia, requiring incision and drainage, and one in a metacarpal, which healed spontaneously with antibiotic therapy alone.

PREMATURE INFANTS

The six premature infants in this small series are of particular interest. Their records contain detailed observations of their conditions charted several times a day, from the time of entry into the special premature nursery until discharge. A tabulation of the pertinent data is given in Table I.

Despite opportunities for close observation by nurses, house staff, and attending pediatricians, there was a delay of from three to eight days (average, five days) between the onset of hip-joint suppuration and the start of treatment by surgical drainage (three cases) or aspiration and instillation of penicillin (three cases). This delay is tabulated in Table II.

Delay in diagnosis was due to failure to recognize the early clinical manifestations of septicemia and of hip-joint suppuration. Apparently unheeded were many observations recorded in the nurses' notes: "Cries when moved. Irritable. Appears in distress when legs are touched or moved."; "Large amount of edema of thigh, leg, and foot. Some discoloration of foot."; "Cries when legs are straightened. Keeps them up at all times in fetal position."; "Baby screaming as though in pain while being handled."; "Left foot appears edematous. Appears to have

pain in lower extremities."; and, "Edema in right buttock and leg—also foot. Cries when touched or moved."

In four infants the discovery of suppuration and pathological dislocation of the hip, extremely rapid in development, led to the diagnosis of septicemia, which was then confirmed by blood culture.

It appears from a study of this small group of premature infants that the method of local hip treatment is less important than the speed with which the diagnosis is made and the treatment started. A delay of four days—during which time the cartilaginous femoral head is bathed in staphylococcus pus under tension—can result in disappearance of the head regardless of the type of local treatment ultimately used.

FULL-TERM INFANTS

Of the nine full-term infants in the neonatal period, infection developed in only one while in the hospital nursery. Infection developed in the remaining eight infants at home, and they were brought back to the hospital for treatment. The exact time of onset of the septicemia could not be determined from questioning of the mothers. An approximate time of onset of hip-joint infection was obtained in eight of the nine infants, varying from one to seven days (average, five days) prior to the time the diagnosis was established.

The study of the full-term infants shows that four of six hip-joint infections —hemolytic *Staphylococcus aureus*—resulted in disappearance of the femoral head (Table III). One femoral head survived despite a delay of sixteen days from onset of infection to surgical drainage. One infant could not be traced and the status of the femoral head is not known. There was a span of from four to sixteen days from onset of hip infection to treatment by surgical drainage or by aspiration and penicillin instillations. The poor results in this group are similar to those in the premature infants and it again may be assumed to be due to delay in diagnosis and treatment. Delay is disastrous.

SUMMARY AND CONCLUSIONS

Suppurative arthritis of the hip is a serious lesion in premature and full-term infants in the neonatal period. Septicemia, the precursor to hip-joint suppuration, is difficult to recognize at this age. The clinical manifestations of septicemia in the neonatal period are reviewed.

Hemolytic *Staphylococcus aureus* was the most common organism recovered from the blood stream and the hip joint. It was found in five of the six premature infants and in seven of the nine full-term infants, including one with bilateral hip involvement.

In fifteen hips hematogenous osteomyelitis of the intracapsular portion of the femur (eleven hips) and of the ilium (four hips) was the source of the joint pus. In the one hip that showed no focus of involvement in the bone, the hemolytic streptococcus infection may have reached the joint by synovial implantation of organisms.

Subluxation or pathological dislocation of the hip was seen on roentgenograms in all the infants in this group.

In twelve hip joints infected with hemolytic *Staphylococcus aureus*, eight femoral heads disappeared. In all eight there was a delay of from four to sixteen days from the onset of hip-joint suppuration to local treatment of the hip, either by surgical drainage or by repeated aspiration and instillations of penicillin.

The most important factors in saving the cartilaginous femoral heads in infected hip joints in the neonatal period are early diagnosis and prompt evacuation of pus, preferably by surgical incision and drainage.

Systemic antibiotics proved to be effective in controlling septicemia and osteomyelitis, but they were unable to sterilize the infected hip joints. Antibiotics could not take the place of surgical drainage.

REFERENCES

1. Blanche, D. W.: Osteomyelitis in Infants. J. Bone and Joint Surg., **34-A**: 71-85, Jan. 1952.
2. Green, W. T., and Shannon, J. G.: Osteomyelitis of Infants. A Disease Different from Osteomyelitis of Older Children. Arch. Surg., **32**: 462-493, 1936.
3. Nicholson, J. T.: Pyogenic Arthritis with Pathologic Dislocation of the Hip in Infants. J. Am. Med. Assn., **141**: 826-831, 1949.
4. Phemister, D. P.: Changes in the Articular Surfaces in Tuberculous and in Pyogenic Infections of Joints. Am. J. Roentgenol., **12**: 1-14, 1924.
5. Smith, R. T.; Platou, E. S.; and Good, R. A.: Septicemia of the Newborn. Current Status of the Problem. Pediatrics, **17**: 549-575, 1956.
6. Weintraub, D. H.: Personal communication.

DISCUSSION

Dr. Jesse T. Nicholson, Philadelphia, Pennsylvania: This has been a timely presentation of a condition that should be called to our attention about every five years. Acute suppurative arthritis of the hip in infants is not seen frequently, even by those of us who work at children's hospitals. We are, therefore, apt to let down our guard—the instruction of interns, residents, obstetricians, and pediatricians—between cases that occur, on an average, once every nine months.

Dr. Obletz remarked about the lack of febrile reaction in the neonatal infant with septic arthritis. In this respect these infants are like the elderly patient, who, in the presence of an extensive infection, shows no reaction of resistance. It is, therefore, well to emphasize that any infant with diarrhea, lymphadenitis, parotitis, draining umbilicus, cellulitis about an intravenous cut-down, or stitch infection after pyloroplasty or tracheo-esophageal plasty, should be considered to have septicemia, especially if he is difficult to feed, irritable, and anemic. Any infant considered to have *septicemia* must be suspect of having suppurative arthritis or osteomyelitis.

It has not been uncommon to find other joints involved, or osteomyelitis of bones other than those associated with the hip joint. In like manner, I should caution that the surgeon is not to be so absorbed with other septic joints or sites of osteomyelitis that he neglects to remember that daily examination of the hip is necessary to detect septic arthritis.

The appropriate antibiotic to which the bacterial organism is sensitive is not only life saving but joint saving. I have no fixed rule about open drainage of the hip. I have found effective: needle aspiration, saline irrigation, and instillation of an antibiotic, as indicated by sensitivity tests, twice daily or as long as there is material yielding a positive culture aspirated from the joint. Joints are saved by early detection of infection and immobilization, rather than by open drainage. If an abscess is obviously bulging beyond the confines of the joint—a frequent finding if the infection has been of ten or more days' duration—it should be drained. Joints have been observed to be remarkably resistant to infection if they are immobilized in order that the synovial fluid can take over the bacterial management.

I have immobilized hips in a double hip spica in as much abduction as possible, changing casts at intervals of a few days until a full 180-degree relation of the thighs was obtained. The plaster cast was windowed to permit continuation of the indicated aspiration of the joint. The abducted position of the hips was maintained by plaster cast and brace until ossification of the capital epiphysis appeared adequate for weight-bearing. If this failed to develop by the time the child was diaper-broken, a non-weight-bearing, or patent bottom brace with a built-up shoe on the opposite foot, was utilized until the epiphysis and acetabulum had every chance to develop, even if the brace was worn through the thirteenth year. In infants in whom the pathological dislocation had been of many months' standing, open reduction, after a period of traction on the leg, was necessary to replace the proximal end of the femur in the acetabulum.

Arthrodesis prior to the time for the completion of the proximal femoral growth, I believe, is responsible for additional discrepancy in leg length. That this growth continues has been shown in six patients with unilateral involvement with destruction of the capital epiphysis, who were followed into adult life. The discrepancy in the femoral length in these patients was but one and one-quarter to one and one-half inches. This shortening corresponded to the depth of the head on the opposite (normal) side.

May I emphasize again conservative management to preserve the epiphyseal growth, and early reduction of the dislocation to keep the physiological relationship of the proximal epiphysis and the acetabulum.

Dr. Edward L. Compere, Chicago, Illinois: Not even the late J. Albert Key, with his remarkable ability to debunk almost any paper, would find anything serious with which to disagree in the paper presented by Dr. Obletz.

During the 1940's all who were associated with children's hospitals or taking care of the orthopaedic problems of newborn infants, were undoubtedly aware of the fact that, in the later part of the '40's, there was a tremendous decrease, in fact almost a complete absence, of acute osteomyelitis. The most probable explanation for this was that every fever, or even semblance of illness without much fever, was treated by a pediatrician, by the family doctor,

(*Continued on page 143*)

The Effects of Continuous Compression on Living Articular Cartilage

An Experimental Investigation *†

BY ROBERT B. SALTER, M.D., M.S., F.R.C.S.(C), AND PAUL FIELD, M.B., B.S.,
TORONTO, ONTARIO, CANADA

From the Department of Orthopaedic Surgery and the Research Institute of the Hospital for Sick Children, Toronto

The problem that prompted the present investigation arose from clinical observations of the sequelae of immobilization of joints in patients who were receiving various forms of orthopaedic treatment. The transient joint stiffness associated with immobilization of short duration is well known. However, one of us (R.B.S.) made the general observation that, if force was required to place a joint in a given position—hereinafter referred to as a forced position—and if that position was maintained during the period of immobilization, the associated joint stiffness tended to persist and to be followed, eventually, by clinical and roentgenographic evidence of significant joint degeneration. The observation of joint degeneration associated with immobilization of a joint in a forced position was recognized as a problem which merited study.

The first step of the study entailed a search in the literature of relevant knowledge, a review of which will be presented as a background for the ensuing experimental investigations.

A REVIEW OF RELEVANT KNOWLEDGE

In view of the fact that articular disorders rank foremost among crippling conditions of man [4], the literature pertaining to fundamental studies of joints is disproportionately limited and thus indicates the need for more intensive investigation.

I. *Embryology and Anatomy of Articular Cartilage*

Embryologically, all the components of a joint are mesenchymal in origin. The joint cavity may be considered a tissue space lined by a mesenchymal membrane and containing synovial fluid, which is composed of amorphous intercellular substance diluted with tissue fluid [20,21].

The gross characteristics of normal articular cartilage are best studied in the freshly dissected specimen. During childhood, articular cartilage is translucent and bluish; however, in adult life it gradually becomes opaque and yellowish [13]. The cartilage is resilient and its articular surface is smooth, glistening, and slippery. Charnley recently demonstrated that the coefficient of friction between cartilage and cartilage is three times as slippery as ice on ice. Gardner emphasized that during movement the surfaces of synovial joints are incongruent, except for a small area of potential contact.

II. *Histology and Physiology of Articular Cartilage*

Microscopic Structure

The most striking feature of the microscopic appearance of articular cartilage is the preponderance of intercellular substance over cells. The amorphous

* Read at the Annual Meeting of The American Orthopaedic Association, Lake Placid, New York, June 16, 1959.

† This investigation was assisted with funds allocated by the Province of Ontario under the National Health Grant Programme, Department of National Health and Welfare, Ottawa.

component of the intercellular substance is composed of chondroitin sulphate [37]. The formed component consists of collagen fibers that are systematically arranged, presumably to provide both strength and resilience [23]. These fibers arise as coarse bundles from the deepest zone of articular cartilage and extend perpendicularly toward the articular surface between rows of chondrocytes. As they approach the surface, however, they split into smaller bundles which curve to run radially, thereby forming a network of fibers parallel to the articular surface [7].

The chondrocytes, lying in lacunae in the intercellular substance, are arranged in four ill-defined strata [13]: (1) a superficial zone containing small, flattened cells; (2) a transitional zone in which the cells are more nearly round; (3) a deep zone with cells lying in perpendicular rows between bundles of collagen fibers; and (4) a calcified zone, demarcated from the deep zone by a wavy line which stains blue with basic dyes. The calcified zone rests on a thin layer of subchondral lamellar bone to which it is securely attached by interdigitation and by continuity of its fibril system with the fibrils of the underlying bone [13]. Articular cartilage contains no blood vessels, lymphatics, or nerves [4,21].

Nutrition of Articular Cartilage

Since articular cartilage is avascular, the chondrocytes must receive their nourishment by a process of diffusion of tissue fluids through the intercellular substance. Thus, the possible sources of nutrition of articular cartilage are synovial fluid and subchondral blood vessels. Most writers, including Strangeways, Fisher [18], Bauer and associates, Collins, Gardner, and Ham, consider synovial fluid to be the chief source of nutrition of articular cartilage. Two phenomena support this view: (1) the survival of cartilage overlying avascular necrosis of subchondral bone [38]; and (2) the survival and, on occasion, the growth of cartilaginous loose bodies lying free in the synovial fluid of a joint [11,29]. Recent ingenious investigations by Ekholm, employing radioactive gold, and Brodin, employing a fluorescent chemical, reveal that, in addition, some nutrition of articular cartilage may also be derived by diffusion from the subchondral vessels. The nutritive fluid from either source reaches the chondrocytes by diffusion through the intercellular substance of the cartilage, and it was demonstrated by Kroh, König, Bennett and associates, Rouvière, Harrison and associates, and Ekholm [15] that the process of diffusion is enhanced by joint movement.

Synovial Fluid

Synovial fluid is a clear, straw-colored, viscous liquid. It is in diffusion equilibrium with blood plasma and, in this respect, resembles other tissue fluids [4]. The synovial fluid is, therefore, admirably suited for the nutrition of articular cartilage. Synovial fluid also contains a ground substance, mucin, which serves as one means of lubrication of the joint. Barnett demonstrated the importance of normal synovial fluid in the prevention of wear and tear in articular surfaces.

Growth, Maintenance, and Metabolism

During the growing period, the deep layer of articular cartilage provides for growth of the epiphysis, just as the epiphyseal plate provides for growth of the diaphysis [13,21]. During this period, mitotic figures may be observed in the chondrocytes; however, after growth has ceased, mitotic figures are no longer seen and amitosis becomes the sole normal method of maintenance of growth of articular cartilage [16]. The metabolism of cartilage, as indicated by studies of its oxygen consumption, is almost nil [9] and decreases with advancing years [33].

III. *Pathology of Articular Cartilage*

Two aspects of joint pathology, the repair of articular cartilage and the

pathogenesis of degenerative arthritis, are particularly relevant to the present investigation and will be reviewed briefly.

Repair of Articular Cartilage

Although it had long been appreciated that articular cartilage was a relatively inactive tissue with limited ability to regenerate [36], Leidy, in 1849, was the first to demonstrate the failure of healing in experimental fractures of cartilage. Redfern, in 1851, reported that incisions in the articular cartilage of dogs showed little evidence of repair. Fisher, in 1923, noted that defects in the central area of articular cartilage demonstrated no evidence of repair, whereas those near the periphery tended to heal by invasion and metaplasia of adjacent connective tissue. Bennett and associates showed that superficial defects of articular cartilage do not heal, whereas defects extending through the depths of the cartilage into subchondral bone result in an unsatisfactory type of healing by invasion of connective-tissue cells and granulation tissue from the bone marrow. In a subsequent study, Bennett and Bauer found that the repair of articular cartilage is no more rapid or complete in puppies than in older dogs.

Although there are slight differences of opinion concerning details of the type of healing, it is universally agreed that articular cartilage exhibits extremely limited power of repair. Hence, local destruction of articular cartilage represents an irreparable and, therefore, serious lesion.

Pathogenesis of Degenerative Arthritis

The term *degenerative arthritis* will be used in the present paper in preference to the more common synonym *osteo-arthritis* because, as Bauer and Bennett pointed out, the word *degenerative* when used in connection with arthritis accurately describes the changes that occur in this condition.

It has been emphasized that destruction of articular cartilage is the starting point in the evolution of degenerative arthritis, and that all subsequent changes in the various components of the joint are secondary [3,13]. The initial destruction may occur spontaneously with normal wear and tear in aging cartilage, or it may occur prematurely in articular cartilage that has been damaged by a local condition, such as infection or injury. The normal free and smooth movement of the joint becomes disturbed, and this disturbance produces further damage to articular cartilage. The underlying bone responds by regeneration and hypertrophy but the articular cartilage responds by degeneration. Once the process has been initiated, the joint gradually, but steadily, deteriorates with continued use. Thus, local destruction of articular cartilage represents not only an irreparable lesion but also one that may result in the development of degenerative arthritis in the involved joint.

THE HYPOTHESIS AND PURPOSE OF THE PRESENT INVESTIGATION

The study of relevant knowledge concerning articular cartilage provided useful information; however, it failed to provide an explanation to the clinical problem that prompted the present investigation, namely, the joint degeneration associated with immobilization of a joint in a forced position. Therefore, a hypothesis was evolved in an attempt to explain the problem.

It was reasoned that when a joint is placed in a forced position, the two opposing areas of articular cartilage in actual contact in that position would be forcibly compressed against one another; furthermore, during the period of immobilization of a joint in a forced position, the two opposing areas of articular cartilage would be subjected to continuous compression. Since articular cartilage receives its nourishment by diffusion of nutritive fluid through its intercellular substance, and since articular cartilage is resilient, it was further reasoned that continuous compression of an area of cartilage would prevent diffusion of nutritive fluid and might, in this way, result in local destruction of carti-

FIG. 1-A

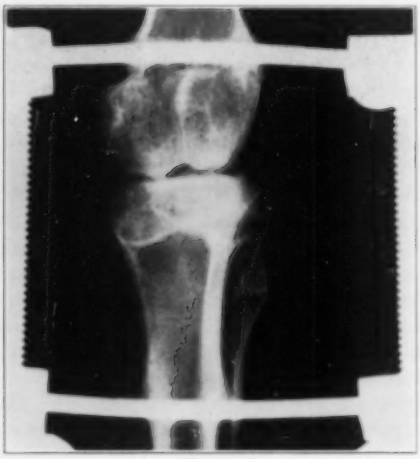


FIG. 1-B

Anteroposterior and oblique roentgenograms of the knee of a rabbit with a compression clamp applied. Note the relatively small area of articular cartilage being compressed.

lage. If this proved to be so, it seemed logical to postulate that such local destruction of articular cartilage might be the starting point in the development of joint degeneration. The evolved hypothesis, therefore, was that the joint degeneration associated with immobilization of a joint in a forced position could be explained on the basis of continuous compression of articular cartilage.

The purpose of the present experimental investigation was to determine the nature and extent of the effects of continuous compression of living articular cartilage in animals as a test of the validity of the aforementioned hypothesis.

No reference to a previous study of this aspect of articular cartilage could be found in the literature.

THE EXPERIMENTAL DESIGN AND METHODS

I. *The Experimental Design*

A preliminary series of experiments was designed to study the effects of relatively prolonged and forceful continuous compression of living articular cartilage in the knee joint of the monkey (Series 1). At the time this preliminary series of experiments was conducted (November 1954) one of us (R.B.S.) was visiting orthopaedic centers in Great Britain as a McLaughlin Travelling Fellow; consequently, the preliminary series was, of necessity, limited in its scope. However, the results of this preliminary study warranted an extension of the investigation. Accordingly, plans were made to study the effects of varying durations of continuous compression of living articular cartilage in the knee joint of the rabbit (Series 2). In an attempt to relate the experimental investigation more closely to the clinical problem, another series of experiments was designed to study the effects, in the rabbit, of the continuous compression of living articular cartilage that resulted from immobilization of a joint in a forced position (Series 3).

II. *The Experimental Methods*

Series 1

The knee joint of the monkey was chosen for study because of its similarity to the knee joint of man with respect to both function and anatomy. This series of experiments was conducted in England at The London Hospital.

Technique: Each monkey was anesthetized by intravenous sodium thiopen-

tone after which one knee was shaved and then prepared with a solution of merthiolate. A stainless-steel pin, four millimeters in diameter, was drilled transversely through the distal metaphysis of the femur in the coronal plane. A second pin, of the same type, was then drilled through the proximal metaphysis of the tibia parallel to the first pin. A Charnley compression clamp was then applied with the knee in extension, and the wing nuts of the clamp were tightened maximally by hand. This method provided forceful compression of the joint surfaces. A cylindrical plaster cast was then applied to maintain the extended position of the knee. The knee joint, in each of four monkeys, was compressed by this method. Two animals were sacrificed at eight weeks, one at nine weeks, and one at twelve weeks.

Controls of Series 1: Two types of control joints were studied: (1) the opposite, mobile knee in each of the four monkeys in the series; and (2) one knee joint in each of three additional monkeys in whom the knee had been immobilized in a plaster cast without compression.

Series 2

The knee joint of the rabbit was chosen for the second series of experiments because of its relative similarity to the knee joint of man with respect to its anatomy and because it was realized that the rabbit was a more practical experimental animal than the monkey. The experiments of Series 2 and Series 3 were conducted at The Research Institute, Hospital for Sick Children, Toronto.

Technique: Each rabbit was anesthetized by intravenous pentobarbitone after which one knee was shaved and then prepared with a solution of green soap. Stainless-steel pins, two millimeters in diameter, and a small specially designed compression clamp were used in this series in the same manner as described in Series 1. Because of the smaller pins and clamp, the compression obtained in this series was less forceful than that obtained in Series 1 (Figs. 1-A and 1-B). A cylindrical plaster cast was then applied to maintain the extended position of the knee. Fifty-seven knee joints were studied in this series, and the animals were sacrificed after periods of compression ranging from two days to nineteen weeks.

Controls of Series 2: Two types of control joints were studied: (1) the opposite, mobile knee in each of fourteen rabbits in the series; and (2) the opposite knee in each of seven rabbits in which stainless-steel pins had been inserted without application of the clamp, but with the knee immobilized in a plaster cast in neutral position.

Series 3

In this series of experiments in the rabbit, continuous compression of two opposing areas of articular cartilage was obtained by immobilizing various joints in a forced position. The particular forced position used for each joint requires description:

The ankle joint was immobilized in a position of forced dorsiflexion with the knee extended. This position was resisted by the calf muscles and produced compression of the anterior margin of the ankle joint. The knee joint was immobilized in some rabbits in a position of forced extension. Since the knee of the rabbit normally extends to only 120 degrees, forced extension of the knee to 180 degrees was resisted by the hamstring muscles and produced compression of the anterior portion of the femoral condyles against the opposing surfaces of the tibial condyles. In other rabbits, the knee was immobilized in a position of forced valgus. This position was resisted by the medial collateral ligament and produced compression of the lateral femoral condyle against the lateral tibial condyle.

Both hip joints were immobilized in a position of forced abduction. This position was resisted by the adductor muscles and produced compression of the

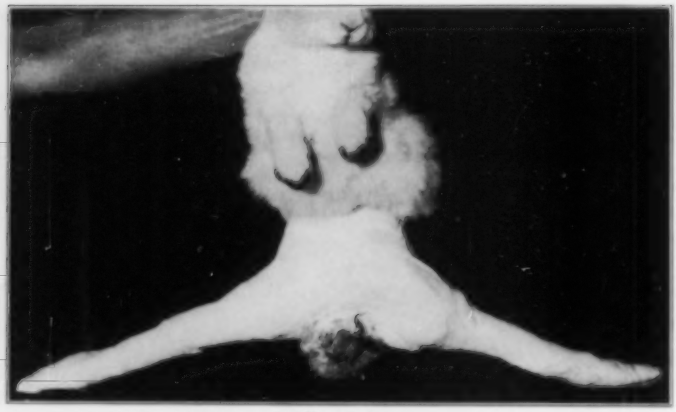


FIG. 2-A

Rabbit with a completed plaster hip spica used for stimultaneous compression of the hip and knee joints in forced positions.

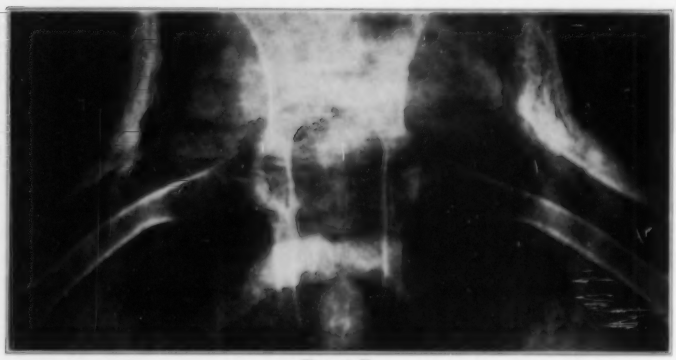


FIG. 2-B

Roentgenogram of rabbit shown in Fig. 2-A.

superolateral surface of the femoral heads against the horseshoe-shaped articular surface of the acetabula. Furthermore, with the hips immobilized in this position, forced extension of the knees, which was resisted by the hamstring muscles, not only produced further compression of the hip joints, but also produced compression of the anterior portion of the knee. Therefore, in some animals both hips and both knees were immobilized in forced positions (Figs. 2-A and 2-B).

Technique: Each rabbit was anesthetized by intravenous pentobarbitone after which the joint under investigation was placed in a forced position and immobilized in a plaster cast. Fifty-two joints were studied in Series 3: four ankles, thirty knees, and eighteen hips. The animals were sacrificed after periods of compression ranging from two days to nineteen weeks.

Controls of Series 3: Two types of control joints were studied: (1) normal, mobile ankle, knee, and hip joints in the rabbit; and (2) twenty joints immobilized in a neutral position. Series 1, 2, and 3 are listed in Table I with respect to the number of joints compressed, the method of compression, and the duration of compression.

III. *Method of Examination of Material*

Careful dissection of the compressed and control joints was performed in order to observe the gross appearance of the articular cartilage and of the other components of the joints. Appropriate blocks of tissue, including cartilage and

FIG. 3

Normal articular cartilage on the femoral condyles of a rabbit. The cartilage is translucent, smooth, and glistening.

subchondral bone, were cut with a fine saw after which they were fixed in neutral formalin, decalcified by sodium citrate and formic acid, and then stained with hematoxylin and eosin.

The gross and microscopic features of normal articular cartilage in the rabbit are shown in Figures 3 and 4.

THE EXPERIMENTAL RESULTS

For the purpose of analysis of the results of the present series of experiments, it was necessary to devise a graded classification based on the gross appearance of the joint surfaces in the freshly dissected specimen. The lesions occurring

FIG. 4

Photomicrograph of normal articular cartilage in the knee of a rabbit. The four strata of chondrocytes and the uniformly basophilic staining of the nuclei of the chondrocytes may be seen. The layer of subchondral bone is thin.

in the articular cartilage could be readily classified as one of three grades. Although the classification was based on gross appearance, each grade was associated with fairly characteristic microscopic features; consequently, both gross and microscopic features will be described for each grade in the classification.

I. *Classification of the Lesions in Articular Cartilage*

Grade I

Gross features: Loss of normal luster and translucency; cartilage yellowish and white in color and soft to touch.

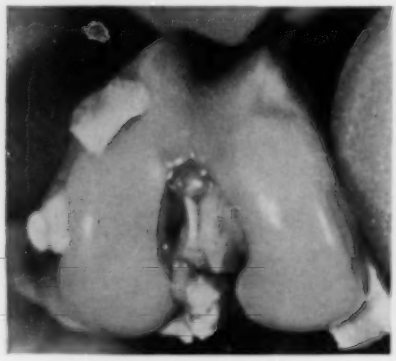


FIG. 5

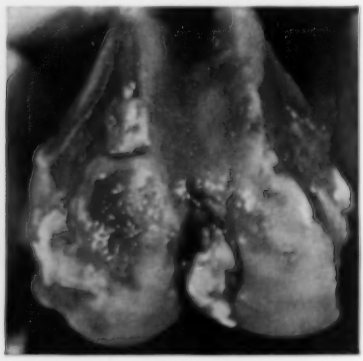


FIG. 7

FIG. 6

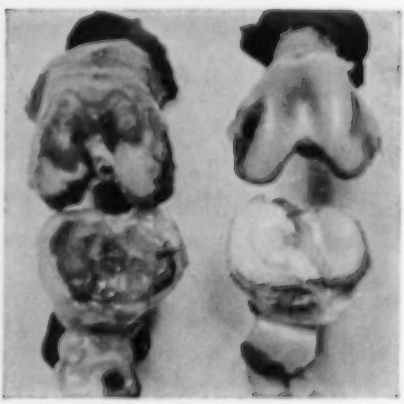


FIG. 8

Fig. 5: Gross appearance of a Grade I lesion on the anterior portion of the medial femoral condyle (right of figure) and on the anterior portion of the lateral condyle. There is loss of normal luster and translucency and the cartilage is yellowish white.

Fig. 6: Gross appearance of a Grade II lesion on both the medial and lateral femoral condyles. There is partial-thickness loss of cartilage. The lesion is deepest in the center, and the peripheral area resembles a Grade I lesion.

Fig. 7: Gross appearance of a Grade III lesion in the anterior portion of the lateral femoral condyle (left of figure): there is full-thickness loss of articular cartilage, exposing subchondral bone in the center of the lesion. (In this knee a Grade II lesion may be seen in the anterior portion of the medial femoral condyle.)

Fig. 8: Series 1. Grade III lesions in opposing areas of the articular surface of the tibia and of the femur (left of figure). Normal control knee of the same monkey is shown at the right.

Microscopic features: Loss of staining power of the nuclei of the chondrocytes in the superficial and transitional zones suggesting necrosis; variable degree of nuclear change in the chondrocytes of the deep zone.

An example of a Grade I lesion is shown in Figures 5 and 9.

Grade II

Gross features: Partial-thickness loss of cartilage or blister formation in center of lesion; changes of Grade I in the periphery.

Microscopic features: Loss of nuclear staining power in the chondrocytes of all layers; disappearance of superficial and transitional zones of cartilage; some hypertrophy of subchondral bone.

An example of a Grade II lesion is shown in Figures 6 and 10.

Grade III

Gross features: Full-thickness loss of cartilage exposing subchondral bone in center of lesion; less marked changes in the periphery.

FIG. 9

Photomicrograph of a Grade I lesion. The loss of staining power of the nuclei of the chondrocytes in the superficial and transitional zones suggests necrosis. There are variable nuclear changes in the chondrocytes of the deep zone.

Microscopic features: Complete absence of chondrocytes and intercellular substance; marked hypertrophy of subchondral bone.

Examples of Grade III lesions are shown in Figures 7, 8, 11, and 12.

Unclassifiable Joints

Nineteen joints in Series 2 and Series 3 could not be classified satisfactorily because of complications which included vascular insufficiency of the limb, septic arthritis, and other conditions. The septic arthritis was considered to be due to

FIG. 10

Photomicrograph of a Grade II lesion showing: loss of nuclear staining power in the chondrocytes of all layers; disappearance of superficial and transitional zones; and hypertrophy of subchondral bone. The edge of the lesion is seen at the left of the figure.

extension of pinprick infection. It was felt that the vascular insufficiency resulted from the constriction of tight plaster casts.

II. *Additional Findings*

Those joints subjected to continuous compression were invariably much stiffer than the joints immobilized without compression, and the degree of stiffness roughly paralleled the severity of the joint lesion. Intra-articular adhesions were almost always present between the compressed articular surfaces at the edge of

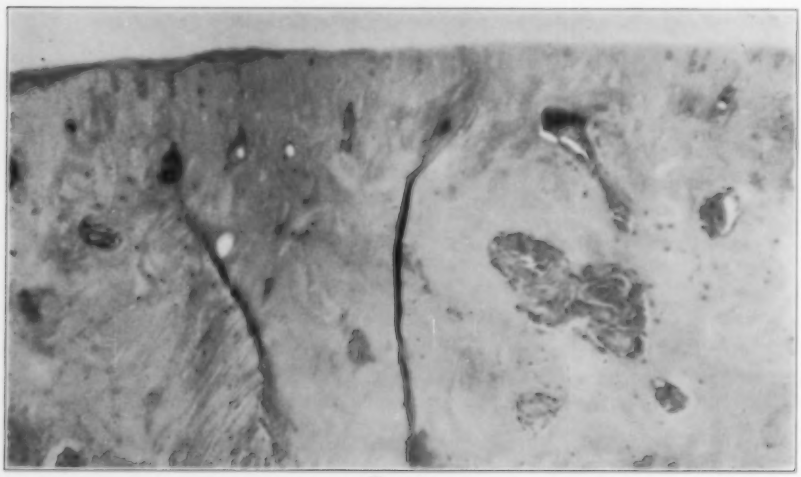


FIG. 11

Photomicrograph of a Grade III lesion. There is complete absence of cartilage on the articular surface and marked hypertrophy of subchondral bone.

FIG. 12

Gross appearance of Grade III lesions on the articular surface of a femoral head. There is full-thickness loss of cartilage, exposing bone in the center of the lesions.

the area of pressure necrosis. Hyperemia was frequently noted in both the synovial membrane and the subchondral bone.

III. *Analysis of the Results*

Lesions in the articular cartilage were observed in all of the eighty-seven classifiable joints subjected to continuous compression for six days or longer. Furthermore, the area of the lesions on the two opposing surfaces of each joint was consistently limited to the area of the joint surfaces that had been in actual contact during the period of compression; this area is referred to hereinafter as the pressure area. Of the seven classifiable joints subjected to continuous compression for less than six days, four exhibited lesions. The shortest period of continuous compression that produced a demonstrable lesion in articular cartilage was three days.

Results in Series 1 (Compression of the Knee of the Monkey by Clamp)

Each of the four knees showed a Grade III lesion. The control knees immobilized without compression were indistinguishable from normal, apart from slight stiffness.

Results in Series 1 (Compression of the Knee of the Monkey by Clamp)

This series consisted of fifty-seven joints and the analysis, based on the duration of continuous compression, is depicted in graphic form in Chart I.

Group A (two to fourteen days): Nine of the seventeen joints were Grade I; four joints were Grade II; and one joint was Grade III. There was no change in two joints compressed for less than three days. One joint was unclassifiable.

Group B (two to six weeks): Eleven of the fifteen joints were Grade II; two joints were Grade I; and two joints were Grade III.

Group C (over six weeks): Nineteen of the twenty-five joints were Grade III; two joints were Grade II; and two joints were Grade I. Two joints were unclassifiable.

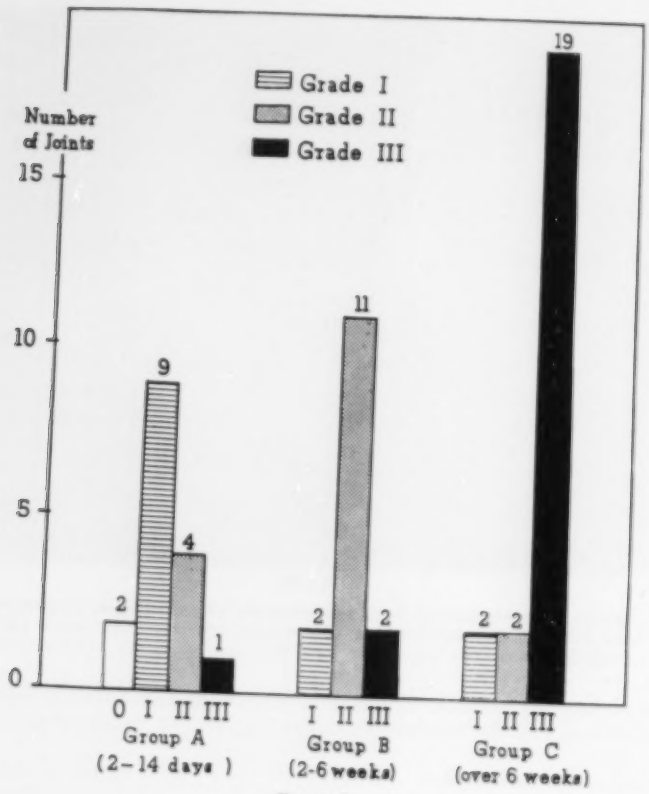


CHART I

Series 2. Analysis of changes in fifty-four classifiable joints compressed by clamp.

Controls of Series 2: No significant abnormality.

Results in Series 3 (Compression of a Joint of the Rabbit by Immobilization in a Forced Position)

This series consisted of fifty-two joints and the analysis, based on the duration of continuous compression, is depicted in graphic form in Chart II.

Group D (two to fourteen days): Five of the nineteen joints were Grade I; three joints were Grade II; there was no change in one joint compressed less than six days; and ten joints were unclassifiable (mostly because of vascular insufficiency of the limb).

Group E (two to six weeks): Nine of the twenty-one joints were Grade I; five joints were Grade II; three joints were Grade III; and four joints were unclassifiable.

Group F (over six weeks): Seven of the twelve joints were Grade III; three joints were Grade II; and two joints were unclassifiable.

Controls of Series 3: No significant abnormality.

DISCUSSION OF RESULTS

It is proposed, in this section, to consider the nature, pathogenesis, and pathological significance of the lesion that has been produced in articular cartilage, and also to discuss a principle that has been formulated as a result of the present investigation.

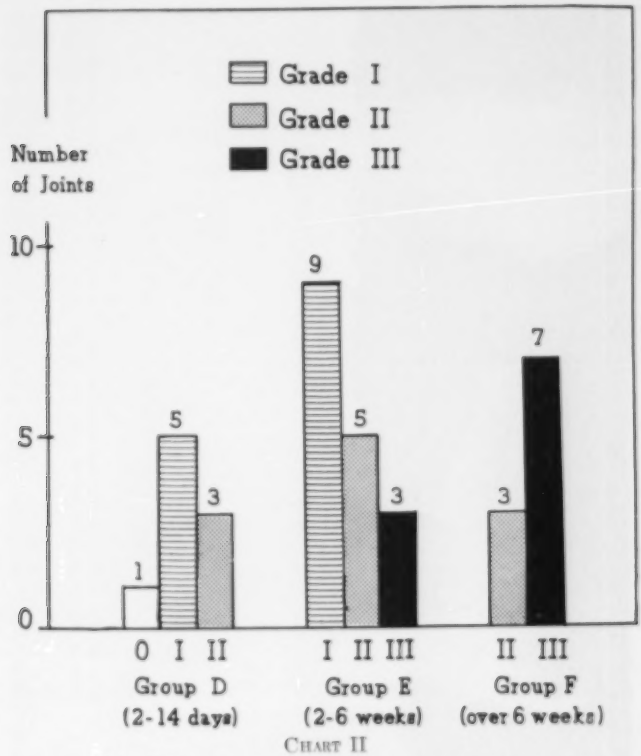


CHART II

Series 3. Analysis of changes in thirty-six classifiable joints compressed by forced position.

I. *Nature and Designation of the Lesion*

The gross and microscopic features of the lesions under consideration reveal that the nature of the pathological change is necrosis of articular cartilage. Anderson defined necrosis as "circumscribed cell or tissue death within the living body", and his description of the morphological features of necrosis could be applied to each of the lesions studied in the present experiments. The Grade I lesions represent necrosis of the superficial and transitional zones of articular cartilage. The Grade II lesions represent necrosis of all zones with liquefaction of the superficial and transitional zones. Grade III lesions represent necrosis of all zones over a larger area with liquefaction of the full thickness of cartilage.

Since the lesions in the present series of experiments were limited to the pressure area of articular cartilage, the designation, *pressure necrosis of cartilage*, is suggested.

II. *Pathogenesis of the Pressure Necrosis of Cartilage*

Continuous compression of articular cartilage for less than three days did not result in pressure necrosis of cartilage, and this indicates that the articular lesions were not produced by simple mechanical destruction. Our concept of the pathogenesis of the pressure necrosis of cartilage is that the continuous compression of the resilient cartilage not only prevented synovial fluid from reaching the surface of the pressure area, but also prevented diffusion of nutritive fluids through the intercellular substance of the cartilage in this area. As a result, the chondrocytes died, the intercellular substance became disorganized, and, finally, both components of the articular cartilage liquefied and disappeared.

FIG. 13

Roentgenograms of the foot and ankle of a boy, fourteen years old. Resistant equinus deformity secondary to Volkmann's ischemic contracture of the calf muscles developed when the boy was seven years old. Clinically, the ankle joint was considered to be otherwise normal prior to passive correction of the equinus deformity. During the ensuing seven years the deformity was passively corrected on four occasions (with the patient under anesthesia). On each occasion the ankle was immobilized in the forced position of dorsiflexion, which was resisted by the contracted calf muscles. Clinically, the foot and ankle were stiff, and movement in the latter was painful. Note the roentgenographic evidence of degeneration in the ankle, subtalar, and mid-tarsal joints. Note, also, the abnormal shape of the talus and the flattening of the ankle joint.

III. *Pathological Significance of Pressure Necrosis of Articular Cartilage*

The possible pathological sequelae of an area of pressure necrosis of articular cartilage in a living animal is, at present, a matter of speculation. However, the pathological significance of this lesion with respect to its reversibility, its effect on future growth of the epiphysis, and its relation to the subsequent development of degenerative arthritis, merit consideration. Pressure necrosis of articular cartilage is probably an irreparable lesion and may interfere with growth of the epiphysis. It may also act as the starting point in the development of degenerative arthritis. Further experiments are being conducted at present to study these two aspects of pressure necrosis of cartilage.

IV. *A Principle Relating to Articular Cartilage*

The results of the present experiments indicate that, in the monkey and in the rabbit, continuous compression of a joint for six days or longer causes pressure necrosis of articular cartilage. Furthermore, the results in Series 3 reveal that, in the rabbit, simple immobilization of a joint in a forced position for six days or

FIG. 14-A

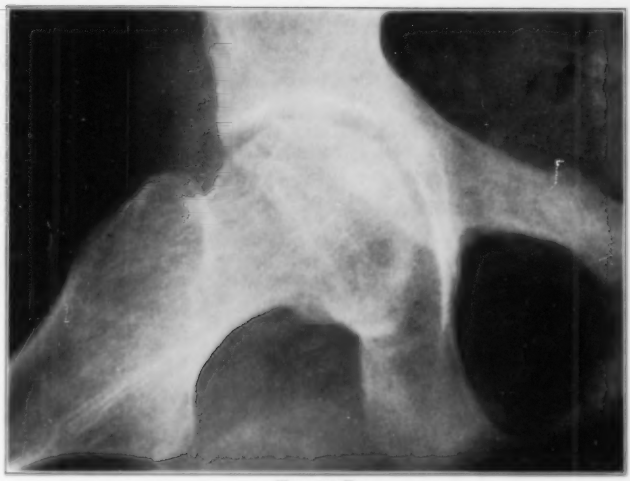


FIG. 14-B

Figs. 14-A and 14-B: Roentgenograms of the hip joints of a fourteen-year-old girl, showing degenerative changes in the right hip. Two years previously, during the treatment of an idiopathic scoliosis, the patient's right hip joint had been immobilized for six months in a position of forced abduction. Two years later the right hip joint was stiff and painful, whereas before the period of immobilization in a forced position it was normal both clinically and roentgenographically. The only explanation that can be offered for the changes in this patient's hip joint is that they are the result of continuous compression of the articular cartilage.

longer provides sufficient compression to cause pressure necrosis of cartilage. Thus, a principle relating to articular cartilage and formulated as a result of the present investigation may be stated: Continuous compression of opposing joint surfaces, whether produced by a mechanical instrument or by simple immobilization of the joint in a forced position, causes pressure necrosis of articular cartilage within six days.

The applicability of this principle to human articular cartilage merits discussion. There is no direct experimental evidence in man—nor is there likely to be—to settle this question unequivocally. It is necessary, therefore, to examine indirect evidence. Other investigators have demonstrated the similarity of articular cartilage in various animals to articular cartilage in man with respect to embryology, anatomy, histology, physiology, and pathology. Therefore, it is probable that, whether continuous compression to articular cartilage is applied to the animal or to the human being, the effects would be the same. When human articular

TABLE I

SERIES OF EXPERIMENTS ON ONE HUNDRED AND THIRTEEN KNEE JOINTS

Experiment	Animal Used	No. of Knee Joints Compressed*	Method of Compression	Duration of Compression
Series 1	Monkey	(4)	Clamp	
		2		8 weeks
		1		9 weeks
		1		12 weeks
Series 2	Rabbit	(57)	Clamp	
Group A		17		2 to 14 days
Group B		15		2 to 6 weeks
Group C		25		Over 6 weeks
Series 3	Rabbit	(52)	Immobilization in a forced position	
Group D		19		2 to 14 days
Group E		21		2 to 6 weeks
Group F		12		Over 6 weeks

*The total number in each series is enclosed in parentheses.

cartilage is subjected to continuous compression by immobilization of a joint in a forced position, the observed effect is eventual joint degeneration, as previously mentioned. This clinical observation lends support to the assumption that the principle is applicable to human articular cartilage.

RELATION OF THE PRESENT INVESTIGATION TO CLINICAL PROBLEMS

This section is based on the assumption that the principle relating to articular cartilage applies in man—an assumption implied by the present experiments and reinforced by clinical observations.

I. *Clinical Significance of Pressure Necrosis of Articular Cartilage*

A comparison may be made between pressure necrosis of articular cartilage and pressure necrosis of skin. It has long been known that continuous compression of skin between an internal bone prominence and a firm external surface causes pressure necrosis of skin. The resultant pressure sore is on an external surface and is painful; it is, therefore, readily detected. It has now been shown that continuous compression of two articular surfaces causes pressure necrosis of the articular cartilage. By contrast, however, the resultant pressure defect in cartilage is both insensitive and internal. Thus, the development of pressure necrosis is a deceivingly painless and occult process; it is, therefore, not readily detected. Furthermore, established pressure necrosis of articular cartilage is probably an irreparable lesion which may act as the starting point in the development of degenerative arthritis in the involved joint. Thus, the prevention of this previously undefined lesion becomes of clinical significance.

II. *Possible Clinical Applications of the Principle*

Immobilization of a joint, or joints, is an essential component of treatment in many conditions of the trunk and extremities. In many instances, the joint is immobilized in a neutral position with no abnormal force acting on it. However, in other instances, the joint may be immobilized in what has been described in this paper as a forced position.

Clinical Situations Associated with Joint Degeneration

It is appropriate to indicate the clinical situations in which we have actually observed clinical and roentgenographic evidence of joint degeneration after immobilization of that joint in a forced position. (In our study we observed: ten

cases of club-foot deformity; two cases of cerebral palsy; one case of poliomyelitis; two cases of Volkmann's contracture; two cases of rheumatoid arthritis; two cases of myodystrophia foetalis; three cases of congenital dislocation of the hip; and one case of paralytic dislocation of the hip.)

The clinical situations may be conveniently discussed under four headings.

Passive correction of a resistant joint deformity: In this situation, passive correction of the deformity is resisted by tight periarticular structures including joint capsule, ligaments, and muscles, or by the abnormal shape of the articular surfaces, or both. Thus, the position of correction, particularly if it has been obtained under anesthesia, represents a forced position, and immobilization of the joint in this position produces continuous compression of joint surfaces:

1. Equinovarus deformity: club-foot, especially of the severe type.

2. Equinus deformity of the ankle: cerebral palsy, poliomyelitis, and Volkmann's contracture (Fig. 13).

3. Flexion deformity of the knee: cerebral palsy, rheumatoid arthritis, and myodystrophia foetalis.

4. Adduction deformity of the hip: congenital and paralytic dislocation of the hip.

Postoperative immobilization of a joint: After certain reconstructive operations such as tendon transfer, peripheral-nerve suture, and cross-leg pedicle skin graft, a joint or joints may be immobilized in a forced position in an attempt to relieve tension on freshly sutured tissues. Excessive abduction of the lower extremity in a scoliosis plaster represents immobilization of the hip in a forced position (Figs. 14-A and 14-B). (In our study we observed: one case of tendon transfer; one case of peripheral-nerve suture; and one case of cross-leg pedicle skin graft.)

Immobilization of a joint in fracture treatment: After reduction of fractures near the ends of long bones, the limb may be immobilized with the joint in a forced position in an attempt to maintain the reduction of the fracture:

1. Supracondylar fractures of the humerus.

2. Fracture-separation of the distal femoral epiphysis.

3. Intertrochanteric fractures of the hip: the well-leg traction method of treatment produces continuous compression of the knee and ankle joints of the normal leg. (In our study we observed: three cases of supracondylar fracture of the humerus; two cases of separation of the distal femoral epiphysis; and one case of well-leg traction.)

Operations on bones and joints: Certain operations involve changing the relative positions of joints and produce compression of articular surfaces:

1. Surgical leg-lengthening: distraction of the osteotomized bone is resisted by the long muscles and is associated with compression of the joint at each end of the bone.

2. Reduction of long-standing congenital dislocation of the hip: if the femoral head is reduced into the acetabulum with force against the resistance of the shortened muscles, compression of the hip joint ensues.

3. Shelf operation for congenital dislocation of the hip: this procedure alters the congruity of the hip joint and compresses the roof of the acetabulum against the superior surface of the femoral head. During the period of postoperative immobilization, this forced position is maintained. (In our study we observed: two cases of surgical leg-lengthening; three cases of reduction of long-standing congenital dislocation of the hip; and two cases of shelf operation.)

III. *A Clinical Suggestion*

The principle formulated in this paper provides an explanation of joint degeneration that we observed in the aforementioned clinical situations. It is suggested that such joint degeneration might be prevented by avoiding immobilization of joints in forced positions.

SUMMARY AND CONCLUSIONS

The experimental investigation of the effects of continuous compression of living articular cartilage was prompted by our clinical observation of joint degeneration associated with immobilization of a joint in a forced position. This observation was recognized as a problem, the explanation of which could not be found in a study of the relevant literature.

The hypothesis that was evolved, in an attempt to explain the problem, was that the joint degeneration under consideration could be explained on the basis of continuous compression of articular cartilage. As a test of the validity of this hypothesis, experiments were designed and conducted to study the effects of continuous compression of living articular cartilage in the monkey and in the rabbit. The compression was produced by a clamp in some animals and by simple immobilization of a joint in a forced position in others; 113 compressed joints were studied. No record could be found of any previous investigation of this aspect of articular cartilage. The results of the present experiments may be summarized as follows:

1. Lesions were observed in the compressed area of articular cartilage in all of the eighty-seven classifiable joints subjected to continuous compression for six days or longer. The lesions varied in extent from superficial necrosis to loss of the full thickness of articular cartilage.

2. The lesions produced by simple immobilization of a joint in a forced position were similar in nature to those produced by clamp compression.

3. The extent of the lesion varied directly with duration of continuous compression.

The articular lesion has been designated *pressure necrosis of cartilage*, and it has been concluded that it is the result of interference with the diffusion of nutritive fluid through the intercellular substance of the cartilage. Pressure necrosis of cartilage is probably an irreparable lesion and may interfere with growth of the epiphysis. It may also act as the starting point in the development of degenerative arthritis.

As a result of the present experiments a principle relating to articular cartilage has been formulated and may now be stated: Continuous compression of opposing joint surfaces, whether produced by a mechanical instrument, or by simple immobilization of the joint in a forced position, causes pressure necrosis of articular cartilage within six days. The similarity of articular cartilage in animals to articular cartilage in man and the observation of joint degeneration associated with immobilization of human joints in forced positions provide indirect evidence in support of the applicability of the principle to articular cartilage in man.

Clinically, the development of pressure necrosis of articular cartilage is difficult to detect because it is both painless and occult; its prevention, therefore, is of considerable importance. Certain clinical situations in which we observed joint degeneration in association with immobilization of a joint in a forced position serve to relate the present investigation to clinical problems.

Finally, it is concluded that the principle formulated in this paper provides an explanation of the joint degeneration under consideration, and the suggestion is made that such joint degeneration might be prevented by avoiding immobilization of joints in forced positions.

REFERENCES

1. Anderson, W. A. D.: Pathology, p. 87. Ed. 3. St. Louis, The C. V. Mosby Co., 1957.
2. Barnett, C. H.: Wear and Tear in Joints. An Experimental Study. J. Bone and Joint Surg., **38-B**: 567-575, May 1956.
3. Bauer, Walter, and Bennett, G. A.: Experimental and Pathological Studies in the Degenerative Type of Arthritis. J. Bone and Joint Surg., **18**: 1-18, Jan. 1936.
4. Bauer, Walter; Ropes, M. W.; and Waine, Hans: The Physiology of Articular Structures. Physiol. Rev., **20**: 272-312, 1940.
5. Bennett, G. A., and Bauer, Walter: Further Studies Concerning the Repair of Articular Cartilage in Dog Joints. J. Bone and Joint Surg., **17**: 141-150, Jan. 1935.

6. Bennett, G. A.; Bauer, Walter; and Maddock, S. J.: A Study of the Repair of Articular Cartilage and the Reaction of Normal Joints of Adult Dogs to Surgically Created Defects of Articular Cartilage, "Joint Mice" and Patellar Displacement. Am. J. Pathol., **8**: 499-524, 1932.
7. Benninghoff, A: Cited by Collins[18].
8. Brodin, Harald: Paths of Nutrition in Articulate Cartilage and Intervertebral Discs. Acta Orthop. Scandinavica, **24**: 177-183, 1955.
9. Bywaters, E. G. L.: The Metabolism of Joint Tissues. J. Pathol. and Bacteriol., **44**: 247-268, 1937.
10. Charnley, John: The "Slipperiness" of Articular Cartilage. *In* Proceedings of The British Orthopaedic Association. J. Bone and Joint Surg., **37-B**: 164, Feb. 1955.
11. Clark, W. E. LeG.: The Tissues of the Body: An Introduction to the Study of Anatomy. Ed. 2. London, Oxford University Press, 1945.
12. Coggeshall, H. C.: The Synovial Fluid. *In* Comroe's Arthritis and Allied Conditions. Ed. 5. Philadelphia, Lea and Febiger, 1953.
13. Collins, D. H.: The Pathology of Articular and Spinal Diseases, pp. 19-23. London, Edward Arnold and Co., 1949.
14. Ekholm, R.: Articular Cartilage Nutrition. How Radioactive Gold Reaches the Cartilage in Rabbit Knee Joints. Acta Anat., Supplementum 15, 1951.
15. Ekholm, R.: Nutrition of Articular Cartilage. A Radioautographic Study. Acta Anat., **24**: 329-338, 1955.
16. Elliott, H. C.: Studies on Articular Cartilages. I. Growth Mechanisms. Am. J. Anat., **58**: 127-145, 1936.
17. Fisher, A. G. T.: Some Researches into the Physiological Principles Underlying the Treatment of Injuries and Diseases of the Articulations. Lancet, **2**: 541-548, 1923.
18. Fisher, A. G. T.: Chronic (Non-Tuberculous) Arthritis: Pathology and Principles of Modern Treatment. New York, The Macmillan Co., 1929.
19. Gardner, Ernest: Physiology of Movable Joints. Physiol. Rev., **30**: 127-176, 1950.
20. Haines, R. W.: The Development of Joints. J. Anat., **81**: 33-35, 1947.
21. Ham, A. W.: Histology, pp. 328-329. Ed. 3. Philadelphia, J. B. Lippincott Co., 1957.
22. Harrison, M. H. M.; Schajowicz, F.; and Trueta, J.: Osteoarthritis of the Hip: A Study of the Nature and Evolution of the Disease. J. Bone and Joint Surg., **35-B**: 598-626, Nov. 1953.
23. Hollander, J. L.: The Structure and Function of Articular Tissues. *In* Comroe's Arthritis and Allied Conditions. Ed. 5. Philadelphia, Lea and Febiger, 1953.
24. Horvath, S. M., and Hollander, J. L.: Intra-articular Temperature as a Measure of Joint Reaction. J. Clin. Invest., **28**: 469-473, 1949.
25. Keith, Arthur: Menders of the Maimed. The Anatomical and Physiological Principles Underlying the Treatment of Injuries to Muscles, Nerves, Bones and Joints. London, Oxford University Press, 1919.
26. Keith, Arthur: Human Embryology and Morphology. Ed. 6. London, Edward Arnold and Co., 1948.
27. König, Fritz: Über reaktive Vorgänge am Knorpel nach verschiedenen Schädigungen. Arch. f. Klin. Chir., **124**: 1-14, 1923.
28. Kroh, Fritz: Studien über den Bau der Synovialmembran und die Resorption des Gelenkinhaltes unter dem Einflusse variabler-mechanischer Momente. Deutsche Zeitschr. f. Chir., **94**: 215-240, 1908.
29. Landells, J. W.: The Reactions of Injured Human Articular Cartilage. J. Bone and Joint Surg., **39-B**: 548-562, Aug. 1957.
30. Leidy, J.: Cited by Subba Rao[36].
31. Luck, J. V.: Bone and Joint Diseases: Pathology Correlated with Roentgenological and Clinical Features. Springfield, Illinois, Charles C. Thomas, 1950.
32. Redfern, Peter: Cited by Keith[25].
33. Rosenthal, Otto; Bowie, M. A.; and Wagoner, George: Studies in the Metabolism of Articular Cartilage. I. Respiration and Glycolysis of Cartilage in Relation to its Age. J. Cell. and Comp. Physiol., **17**: 221-233, 1941.
34. Rouvière, H.: De l'atrophie des cartilages de revêtement dans les articulations normales immobilisées. Ann. d'Anat. Pathol., **14**: 721-724, 1937.
35. Strangeways, T. S. P.: Observations on the Nutrition of Articular Cartilage. British Med. J., **1**: 661-663, 1920.
36. Subba Rao, K. V.: An Experimental Study of Regeneration in Cartilage. J. Pathol. and Bacteriol., **67**: 455-459, 1954.
37. Sylvén, Bengt: Cartilage and Chondroitin Sulphate. I. The Physiological Role of Chondroitin Sulphate in Cartilage. J. Bone and Joint Surg., **29**: 745-752, July 1947.
38. Tovee, E. B.: Fractures of the Neck of the Femur in Dogs. (Unpublished paper read at the Annual Meeting of The American Orthopaedic Association, June 20, 1953.)

DISCUSSION

Dr. G. W. N. Eggers, Galveston, Texas: Dr. Salter and Dr. Field have demonstrated the results of catastrophic pressure on the cartilage of joints. The clinical inference is that immobilization of joints which results in comparable cartilage pressures is consistently disastrous to the cartilage and to subchondral areas. Immobilization causes fibrous tissue to cover the non-compressed cartilage and damages the cartilage because of loss of synovial nutrition. The consideration of physical forces on cartilaginous bone tissues is one of realistic importance.

(Continued on page 76)

Densities of Bones of White and Negro Skeletons*

BY MILDRED TROTTER, PH.D., GEORGE E. BROMAN, M.D.†, AND ROY R. PETERSON, PH.D., ST. LOUIS, MISSOURI

From the Department of Anatomy, Washington University School of Medicine, St. Louis

It is well known that the total skeletal mass changes with age. Evidence is accumulating to support the belief that after early adult life, through the process of internal remodeling of bones, there is an irregular decline in the weight of the skeleton as the bones become less dense or more porous [10]. Finlay has proposed that this change is the result of a number of factors concomitant with aging, such as the endocrine disturbances associated with gonadal hypofunction, the general decrease in physical activity resulting in diminished skeletal stresses and strains, and, possibly, nutritional deficiency.

The density of bones has been determined by several different methods which include subjective evaluation from inspection of either sectioned bones [9] or roentgenograms [13], comparison between the shadows of the bone and either an ivory [11] or an aluminum [12] wedge on the same roentgenogram, radioactive strontium [5], and microradiograms [2,3]. Most studies have been limited to a given bone or, possibly, several bones from a series of subjects or skeletons. It has been found that different bones, as well as different regions of the same bone, show variations in composition [15] which change with age [8], but the data showing these changes are not in agreement.

In studies in this laboratory bone density is considered to be an expression of the weight of the bone per unit volume $\left(\frac{\text{weight}}{\text{volume}}\right)$ not the specific gravity of bone substance, and the volume is considered to be that which is enclosed by the surface of the bone, including both the bone substance and the marrow and other cavities contained within its surface. Previous studies have shown that the mean densities of cervical vertebrae, lumbar vertebrae, humeri, and femora differ significantly within each sex-race group of American whites and Negroes; that the mean densities of each of these four series of bones differ significantly from one another except in the case of the humeri and femora whose mean densities do not differ significantly; that the mean densities of each of the four bone series differ significantly in the four sex-race groups except for the femoral series in which this was not the case; and that the densities of each series of bones decreased with age at rates not proved to differ from each other in the four sex-race groups [4,17,18].

This paper will extend these previous studies to other series of bones from the same skeletons. The densities of these bones will be compared from the standpoint of sex, race and age, and the reliability of the differences will be determined by statistical analyses.

MATERIAL AND METHODS

The material comprises a series of eighty adult skeletons from forty American white and forty American Negro cadavera assigned to Washington University by the Missouri State Anatomical Board, each racial group consisting of twenty males and twenty females of approximately the same ages. The age range, mean

* Supported by Grant A-1013, United States Public Health Service.
† Summer Research Fellow, 1955-1957.

age, and standard deviation (in years) of the twenty skeletons in each sex-race group are as follows:

		Range	*Mean*	*S.D.*
White	male	42 to 94	64.0	12.8
	female	25 to 91	67.2	17.1
Negro	male	27 to 84	59.6	13.9
	female	33 to 100	60.3	19.8

The details concerning the source and preparation of the skeletons may be found in an earlier report [16]. In addition to the four series of bones already studied the densities of radii, ulnae, tibiae, ribs, thoracic vertebrae, and sacra have been determined.

The bones were weighed (in grams) in a dry, fat-free state, and their volumes were determined (in cubic centimeters) by the displacement of millet seed in containers of the smallest size which would accommodate the bones. The principle and details of the method have been recorded elsewhere for the lumbar vertebrae and femora [4], the humeri [17], and the cervical vertebrae [18]. This same method was employed to determine the volume of the bones in the series reported here, but with certain differences in details. A 500-cubic-centimeter graduated glass cylinder was used for the ulnae and radii and a 4,000-cubic-centimeter graduated cylinder for the thoracic vertebrae and ribs. The volume of the tibiae was measured in a cylinder made from pyrex glass tubing with an inside length of 50.6 centimeters and an inside diameter of 9.3 centimeters. A box with inside measurements of fifteen by twenty by ten centimeters was used for sacra. The thoracic vertebrae were placed in the cylinder simulating the anatomical position, whereas the ribs were placed with their heads down and the sacrum with its pelvic surface down. The measurements of each bone or bone series (ribs) of a pair were made separately and then averaged. All volume determinations were made by the same person (G.E.B.).

The reliability of this method of volume determination was tested for each bone by repeating the procedure on ten pairs or series of bones chosen at random. The average absolute difference in volume between the first and second set of measurements was determined in per cent of the average of the first measurements. This was considered to be the percentage of error of the method and it was found to be 1.0 per cent for the cervical and thoracic vertebrae each, 1.3 per cent for the lumbar vertebrae, 2.2 per cent for the sacra, 2.4 per cent for the humeri, 2.5 per cent for the ulnae, 3.1 per cent for the tibiae, 3.2 per cent for both the ribs and the radii, and 3.5 per cent for the femora.

Other bones of the skeleton (mandible, sternum, scapulae, and hip bones) were also measured but the data were not used because the percentage of error was excessively high, ranging from 4.5 to 7.8 per cent. The irregular contour of the hip bones and of the scapulae, requiring a very large container, the fact that the sternum in many instances had been broken or sawed, and the great variability in the number of teeth and the condition of the alveolar border of the mandible were contributing factors to these errors.

In the statistical analyses of the earlier studies, graphic tests indicated that the distributions of the densities of each of the four bone series did not depart significantly from normality, and, thus, transformation of the densities to logarithms was not indicated. However, when the densities of the ten bone series were analyzed, it became apparent that the bone series with the smaller mean densities had smaller standard deviations and, that, as the means increased, the standard deviations increased. For this reason transformation of the densities to logarithms was suggested. Tukey's test for additivity [1] and Bartlett's chi-square test [1] for homogeneity of variances confirmed the desirability of using the logarithmic transformation when analyzing the ten bone series for each sex-race group. In order to avoid using negative numbers, 1 (one) was added to each logarithm. The

TABLE I

MEANS AND STANDARD DEVIATIONS (S.D.) OF WEIGHT (GM), VOLUME (CM), DENSITY $\left(\frac{\text{WEIGHT}}{\text{VOLUME}}\right)$ AND LOG-DENSITY AND THE GEOMETRIC MEANS OF DENSITIES OF EACH SERIES OF BONES ACCORDING TO RACE AND SEX

Bone Series	Weight		Volume		Density		Log-Density Mean (+1)	S.D.	Geometric Mean
	Mean	S.D.	Mean	S.D.	Mean	S.D.			
White males (20) *									
Cervical v.	54.5	9.3	105.7	12.6	.517	.065	.710	.054	.513
Thoracic v.	131.5	31.4	317.0	52.2	.416	.082	.611	.087	.408
Lumbar v.	111.0	20.4	273.0	54.4	.415	.096	.599	.104	.397
Sacra	64.8	15.2	168.2	3.4	.396	.100	.572	.123	.373
Ribs	102.0	25.3	170.8	41.2	.612	.140	.775	.101	.596
Humeri	113.5	22.7	178.5	20.3	.642	.133	.799	.087	.629
Radii	38.1	6.3	52.1	7.2	.734	.123	.860	.073	.724
Ulnae	47.1	8.2	60.4	7.5	.782	.120	.888	.067	.773
Femora	310.4	54.9	499.8	63.1	.628	.114	.791	.079	.618
Tibiae	180.7	37.5	248.8	37.3	.740	.184	.857	.105	.720
White females (20) *									
Cervical v.	37.6	9.6	86.0	13.8	.440	.102	.632	.100	.428
Thoracic v.	95.7	28.4	249.2	50.0	.387	.104	.570	.109	.372
Lumbar v.	79.8	22.9	208.8	36.3	.380	.080	.570	.088	.372
Sacra	46.6	13.0	134.8	42.1	.360	.094	.542	.114	.348
Ribs	65.3	22.3	105.2	29.9	.638	.133	.786	.132	.610
Humeri	65.5	17.3	116.0	17.9	.566	.114	.745	.085	.556
Radii	20.9	6.1	33.7	5.3	.622	.153	.781	.103	.604
Ulnae	27.0	6.5	42.2	5.1	.640	.130	.798	.083	.628
Femora	200.0	48.7	339.6	66.6	.598	.121	.768	.087	.586
Tibiae	110.8	31.2	175.5	34.2	.636	.169	.789	.114	.615
Negro males (20)									
Cervical v.	59.0	13.2	101.6	14.8	.580	.090	.758	.068	.573
Thoracic v.	156.1	35.9	325.3	57.6	.482	.082	.676	.075	.474
Lumbar v.	135.4	40.2	277.0	60.7	.483	.097	.676	.082	.474
Sacra	72.7	15.4	152.8	30.5	.482	.089	.676	.079	.474
Ribs	126.3	24.8	190.9	46.8	.684	.145	.825	.095	.668
Humeri	135.9	21.8	189.3	21.9	.718	.102	.852	.060	.711
Radii	47.4	7.5	60.3	7.4	.786	.096	.892	.053	.780
Ulnae	59.6	9.4	70.1	7.3	.850	.103	.926	.055	.843
Femora	356.0	59.8	511.2	72.6	.700	.087	.842	.054	.694
Tibiae	219.7	35.1	276.6	13.8	.802	.126	.898	.077	.790
Negro females (20)									
Cervical v.	46.4	10.1	88.2	14.2	.533	.121	.716	.101	.520
Thoracic v.	112.9	29.4	257.0	46.2	.444	.111	.635	.105	.431
Lumbar v.	101.9	24.6	229.0	42.1	.458	.141	.644	.120	.441
Sacra	56.9	15.5	129.8	27.4	.455	.154	.636	.142	.432
Ribs	84.6	20.9	138.1	13.3	.675	.269	.797	.174	.626
Humeri	80.8	13.9	129.3	22.4	.640	.139	.796	.097	.625
Radii	28.0	5.1	40.3	6.2	.706	.153	.839	.098	.690
Ulnae	36.3	6.9	50.5	6.6	.723	.133	.852	.084	.710
Femora	235.6	44.2	368.0	59.4	.652	.129	.806	.091	.639
Tibiae	139.3	34.6	213.2	38.1	.662	.171	.806	.117	.640

*In the white male series the cervical vertebrae were not available in two skeletons and the sacrum in one skeleton. In the white female series the thoracic vertebrae were not available in one skeleton. For all except one series of cervical vertebrae substitute values were derived by the missing plot technique[14]; for this series of cervical vertebrae a value based on a skeleton of comparable age, sex, and race was substituted.

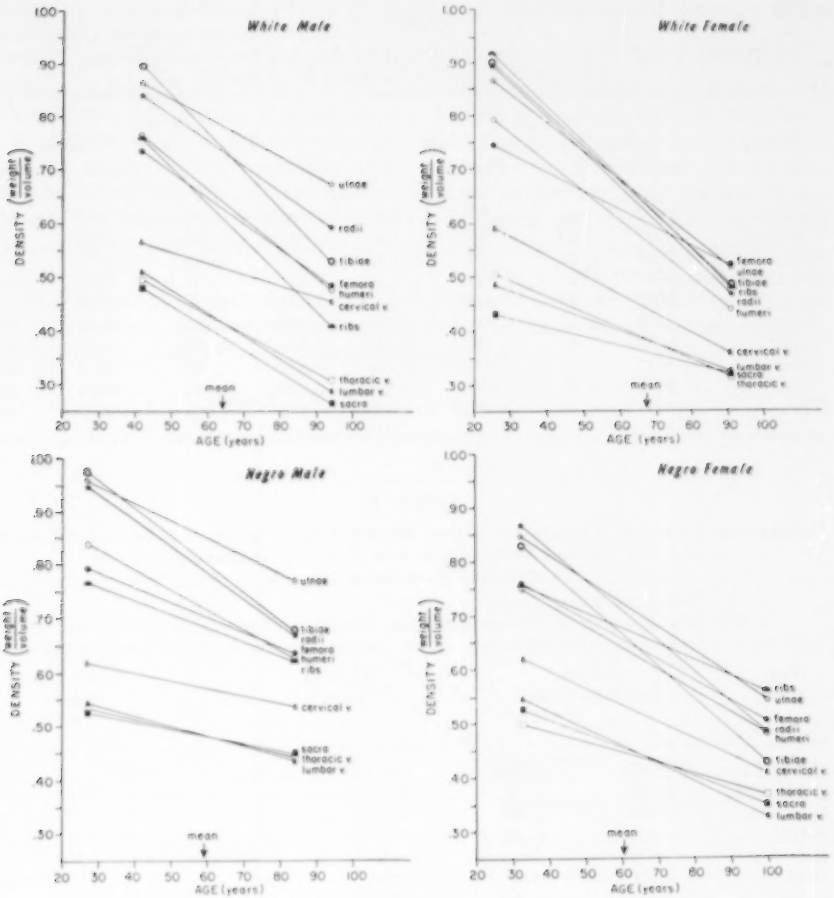


CHART I

Regression lines of density plotted against age of ten series of bones.

anti-logarithm of the mean log-density is the geometric mean of the densities.

RESULTS

The means and standard deviations of weight, volume, density, and log-density, and the geometric means of the density of each series of bones are presented in Table I according to sex and race. Inspection of the data shows that the arithmetic and geometric means of the densities for each of the ten bone series are higher in the Negroes than in the whites of the same sex and also higher in the males than in the females of the same race (except for the ribs in whites).

Effect of Age on Mean Density of Each Bone Series within Each Sex-Race Group

When the densities of each bone series of each sex-race group are plotted against age, it is found that the densities decrease with age, although not significantly in each case. The regression lines are shown in Chart I. The probability that each slope differs from zero depends on the variability among the twenty densities from which each slope is determined and, therefore, differs among slopes which are approximately the same (Table II). Scattergrams were shown in our earlier reports for lumbar vertebrae and femora [4], for humeri [17], and for cervical vertebrae [18]. When the slopes are calculated by means of the logarithmic trans-

TABLE II

REGRESSION COEFFICIENTS (SLOPES) OF DENSITY ON AGE AND PROBABILITY LEVELS (P) FOR EACH BONE SERIES OF EACH SEX-RACE GROUP

Bone Series	Male				Female			
	White		Negro		White		Negro	
	Slope	P	Slope	P	Slope	P	Slope	P
Cervical v.	−.0021	n.s.	−.0010	n.s.	−.0036	<.01	−.0032	<.05
Thoracic v.	−.0037	<.01	−.0016	n.s.	−.0029	<.05	−.0020	n.s.
Lumbar v.	−.0043	<.01	−.0019	n.s.	−.0025	<.05	−.0033	<.05
Sacra	−.0043	<.05	−.0014	n.s.	−.0017	n.s.	−.0026	n.s.
Ribs	−.0067	<.01	−.0026	n.s.	−.0066	<.01	−.0030	n.s.
Humeri	−.0055	<.01	−.0038	<.05	−.0054	<.001	−.0040	<.01
Radii	−.0048	<.05	−.0049	<.001	−.0065	<.001	−.0057	<.001
Ulnae	−.0037	n.s.	−.0033	<.05	−.0054	<.001	−.0045	<.001
Femora	−.0049	<.05	−.0029	<.05	−.0035	<.05	−.0039	<.01
Tibiae	−.0071	<.05	−.0052	<.01	−.0063	<.01	−.0060	<.001

TABLE III

PROBABILITY LEVELS OF DIFFERENCES AMONG MEANS OF LOG-DENSITIES OF SEX-RACE GROUPS AND OF DIFFERENCES ACCORDING TO SEX, RACE AND INTERACTION OF THE TWO* WITHIN EACH BONE SERIES BY:

(I) THE ANALYSIS OF VARIANCE AND
(II) THE ANALYSIS OF COVARIANCE WITH ADJUSTMENT FOR AGE DIFFERENCES

Bone Series	Among Sex-Race Groups		Male vs. Female		White vs. Negro	
	I	II	I	II	I	II
Cervical v.	<.001	<.001	<.01	<.01	<.001	<.01
Thoracic v.	<.01	<.05	n.s.	n.s.	<.01	<.05
Lumbar v.	<.01	<.05	n.s.	n.s.	<.001	<.01
Sacra	<.01	<.01	n.s.	n.s.	<.001	<.01
Ribs	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
Humeri	<.01	<.01	<.01	<.01	<.01	<.05
Radii	<.01	<.01	<.01	<.001	<.05	n.s.
Ulnae	<.001	<.001	<.001	<.001	<.01	<.05
Femora	<.05	n.s.	n.s.	n.s.	<.05	n.s.
Tibiae	<.001	<.01	<.001	<.001	n.s.	n.s.

*Interaction is not significant in any instance.

formation of densities, which is equivalent to fitting an exponential curve to the original data, the probability that each slope differs from zero is the same as when calculated from the original units except in five instances. In four of these instances the slope is more highly significant when the original units are used, and in the other instance the slope is more highly significant when the logarithms are used. For this reason and for the sake of simplicity the slopes in Charts I and II are based on the densities in the original units.

Analysis of Log-Densities of the Bone Series within Each Sex-Race Group

Within each sex-race group the logarithms of the densities of the ten bone series for the twenty skeletons are studied by the analysis of variance for a randomized block incorporating tests of regression of density on age[1] (page 150). In each sex-race group variation attributable to the various skeletons is found to be significantly greater than the experimental error (Table of Variance Ratios[7]). The best straight line fitted to the means of the log-densities of the ten bone series for the twenty skeletons is found to decrease significantly with age

in each sex-race group and there is no evidence that the rate of decrease differs among the ten bone series.

In each sex-race group the means of the log-densities of the ten bone series differ significantly and appear to fall into four natural categories as follows: (1) most dense (radii, ulnae, and tibiae); (2) less dense (humeri, femora, and ribs); (3) still less dense (cervical vertebrae); and (4) least dense (thoracic vertebrae, lumbar vertebrae, and sacra). Analysis of the ten means within each sex-race group as four subseries means shows that most of the variation among the ten means is attributable to these four subseries means and that variation among means within subseries is no more than may be expected by the experimental error. Comparison by Tukey's method [14] of differences in all combinations of any two bones within each sex-race group verified this, and gave the further information that for the two female groups differences in all combinations of two bones within the combined subseries one and two are not significant.

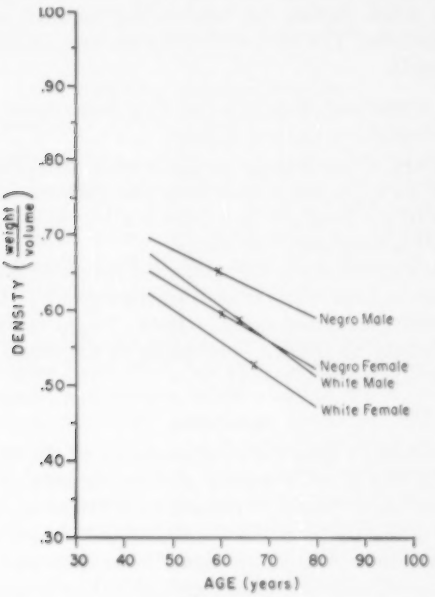


CHART II

Regression lines based on combined slopes of ten series of bones in each sex-race group. The mean density of the ten series is indicated by x at the mean age of each group.

Differences among Means of Log-Densities of Sex-Race Groups within Each Bone Series

The log-densities among the four sex-race groups for each bone series were compared by the analysis of variance for factorial arrangement of sex-race groups according to the model used in the previous studies [4,17,18] in which the variation among the four sex-race groups is segregated into the portion attributable to sex, to race, and to interaction of sex and race.

The differences among the rib series of the four sex-race groups are not significant. The differences for the remaining nine series vary from being highly significant in cervical vertebrae, ulnae, and tibiae ($P < .001$), and in thoracic vertebrae, lumbar vertebrae, sacra, humeri, and radii ($P < .01$) to being significant in the femora ($P < .05$). The densities of the cervical vertebrae, humeri, radii, and ulnae differ significantly between males and females and between whites and Ne-

groes; whereas the densities of the tibiae differ only between males and females, and the densities of thoracic vertebrae, lumbar vertebrae, sacra, and femora differ only between whites and Negroes. The densities of the ribs do not differ between either the two sexes or between the two races. The interaction between sex and race is not significant in any instance. A summary of the levels of probability for these comparisons is presented in Table III in the columns headed I.

In order to determine whether differences among the log-densities of each bone series in the four sex-race groups are due to age differences, the analysis of covariance, adjusting for age differences, has been employed. For the thoracic vertebrae, lumbar vertebrae, and tibiae the probability value is increased, and for the femora the difference becomes not significant. In every instance in which race was significant the probability value is increased with the differences among radii and femora becoming not significant. On the other hand, the probability values for sex are either unchanged or decreased. These changes after adjustment for age differences follow from the fact that age differences are greater between races than between sexes. Again, the interaction between sex and race is not significant in any instance. The probability values are summarized in Table III in the columns headed II.

Comparison of the Combined Slopes of the Ten Bone Series in Each Sex-Race Group

The combined slope of log-density on age in each sex-race group is significant when compared with its appropriate error term and the over-all slope representing the combined slopes of the four sex-race groups is highly significant. There is no evidence of divergence among these four slopes.

The straight lines representing the combined slopes calculated from densities in original units of the ten bone series in each sex-race group (white male = —.0047; Negro male = —.0029; white female = —.0044; Negro female = —.0038) pass through the mean density of the ten bone series at the mean age for the twenty skeletons and are illustrated in Chart II.

DISCUSSION

It must be remembered that these comparisons of the densities (amount of bone per unit volume, not specific density of bone substance) of bones from the same skeleton and of bones from individuals of different age, sex, and race, are based on only eighty skeletons, and that these comparisons are made among groups with only twenty skeletons in a group. Also, these skeletons are from approximately the same socioeconomic level and geographical area, and thus do not represent a cross section of the general population. At the same time, it is recognized that the study is unique, not only in the method employed which provides a measurement of density of an entire bone or series of bones, but also in the fact that so many bones from each skeleton have been measured. The method is not one which can be applied to the living, but the statistically analyzed results confirm some widely accepted impressions gained from the living. Therefore, in this study certain additional findings which are at variance with other impressions may be more confidently accepted.

To be expected was the evidence that, in general, bones of the Negro skeleton are denser than bones of the white skeleton and that the long limb bones have a higher density than bones of the vertebral column since the limb bones have a greater proportion of compact to cancellous bone than do vertebrae. It was expected, also, that the density of a given bone or series of bones will be reduced with age. The evidence from four samples, of twenty skeletons each, however, fails to prove that the rate of loss of density varies between the sexes and races for each bone series or among the bone series within each sex-race group. On the contrary, it suggests that the loss occurs at a uniform and parallel rate in most of the bones tested. However, it is possible that larger samples of skeletons would show

different rates of loss of density with age according to the bone, sex, and race, and perhaps non-linear trends.

In addition, the study shows that the densities of the radii, ulnae, and tibiae are the highest of those determined and that they do not differ from each other. The densities of the humeri, femora, and ribs are next highest and they also do not differ from each other. The densities of these six series of bones do not differ from each other in either female group. The superior and inferior limb bones are similar in structure but very different in function from the standpoint of weight-bearing. The ribs, belonging to the axial, as opposed to the appendicular skeleton, might be expected to have a density more closely related to that of the vertebral column than to that of the limb bones. From the standpoint of weight-bearing the segments of the vertebral column might be expected to increase in density from the cervical segment to the sacrum. However, the reverse order was found to exist with the only significant difference occurring between the cervical segment (densest) and the other three segments in the male groups. Thus, it appears that the density of a bone is related more to its structure than to its function.

SUMMARY AND CONCLUSIONS

The densities $\left(\frac{\text{weight}}{\text{volume}}\right)$ of the cervical vertebrae, thoracic vertebrae, lumbar vertebrae, sacra, ribs, humeri, radii, ulnae, femora, and tibiae from eighty adult skeletons were determined. The skeletons were derived equally from American whites and Negroes of both sexes, each group with a wide age range. The weight of the bones was determined with the bones in a dry, fat-free state, and the volumes were measured by displacement of millet seed.

It was found that:

1. In both white and Negro males the mean densities of radii, ulnae, and tibiae did not differ from each other, but they were significantly higher than the densities of ribs, humeri, and femora which did not differ from each other; cervical vertebrae were significantly less dense than the last-named series, but denser than the thoracic vertebrae and lumbar vertebrae and the sacra, which likewise did not differ from each other. In females of both races the mean densities of the five long limb bones and ribs did not differ from each other, but they were significantly higher than the densities of the four segments of the vertebral column which did not differ significantly from each other except that the cervical vertebrae were significantly more dense than the sacra in white females.

2. The density of each bone series in each sex-race group decreased with age although not significantly so in eleven of the forty slopes studied. The combined slope of the densities of the ten bone series in each sex-race group decreased significantly with age. The decline occurred at approximately the same rate in each bone series within each group.

3. After correction for age differences among the four sex-race groups the four mean densities of each bone series (except the ribs and femora) differed significantly. The difference in cervical vertebrae, in humeri, and in ulnae was attributable to sex (male bones denser than female) and to race (Negro bones denser than white). In the radii and in tibiae the difference was attributable to sex only; and in the thoracic vertebrae, in lumbar vertebrae, and in the sacra, to race only.

Thus, from this study of ten series of bones from the same skeletons it was found with only few exceptions that long limb bones and ribs are denser than vertebrae; that cervical vertebrae are denser than the other segments of the column in males, but not significantly so in females; that bones of the Negro skeleton are denser than bones of the white skeleton; and that bones of the male skeleton are denser than bones of the female skeleton. Furthermore, it was suggested by this study that within each sex-race group bones decrease in density with age at a uniform and parallel rate.

NOTE: The statistical methods were selected and carried out by Barbara Bartels Hixon, Lecturer in Biostatistics, Department of Preventive Medicine, whose continued interest and cooperation are acknowledged with gratitude.

REFERENCES

1. BLISS, C. I., and CALHOUN, D. W.: An Outline of Biometry. New Haven, Yale Coop Corp., 1954.
2. BOHATIRCHUK, F.: Some Microradiographical Data on Bone Aging [Abstract]. Anat. Rec., **115**: 286-287, 1953.
3. BOHATIRCHUK, F.: Aging and Osteoarthritis. Canadian Med. Assn. J., **76**: 106-114, 1957.
4. BROMAN, G. E.; TROTTER, MILDRED; and PETERSON, R. R.: The Density of Selected Bones of the Human Skeleton. Am. J. Phys. Anthrop., **16**: 197-211, 1958.
5. EVANS, F. G.; COOLBAUGH, C. C.; and LEBOW, MILTON: An Apparatus for Determining Bone Density by Means of Radioactive Strontium (Sr^{90}). Science, **114**: 182-185, 1951.
6. FINLAY, J. M.: Osteoporosis. Canadian Med. Assn. J., **74**: 823-828, 1956.
7. FISHER, R. A., and YATES, F.: Statistical Tables for Biological, Agricultural and Medical Research. Ed. 5. Edinburgh, Oliver and Boyd Ltd., 1957.
8. GERSHON-COHEN, J.; SCHRAER, HARALD; and BLUMBERG, NATHAN: Bone Density Measurement of Osteoporosis in the Aged. Radiology, **65**: 416-419, 1955.
9. HANSEN, GERHARD: Die Altersbestimmung am proximalen Humerus- und Femurende im Rahmen der Identifizierung menschlicher Skelettreste. Wissenschaft. Zeitschr. der Humboldt-Universitaet, Berlin. Mathemat.-naturwiss., **3(1)**: 1-73, 1953/54.
10. INGALLS, N. W.: Observations on Bone Weights. Am. J. Anat., **48**: 45-98, 1931.
11. MACK, P. B.; BROWN, W. N., JR.; and TRAPP, H. D.: The Quantitative Evaluation of Bone Density. Am. J. Roentgenol., **61**: 808-825, 1949.
12. MAINLAND, DONALD: A Study of Age Differences in the X-Ray Density of Five Bones in the Adult Human Wrist and Hand. J. Gerontol., **12**: 284-291, 1957.
13. SANTE, LEROY: Principles of Roentgenological Interpretation. Ed. 10. Ann Arbor, J. W. Edwards, 1955.
14. SNEDECOR, G. W.: Statistical Methods Applied to Experiments in Agriculture and Biology. Ed. 5. Iowa City, Iowa State College Press, 1956.
15. STROBINO, L. J., and FARR, L. E.: The Relations to Age and Function of Regional Variations in Nitrogen and Ash Content of Bovine Bones. J. Biol. Chem., **178**: 599-609, 1949.
16. TROTTER, MILDRED, and PETERSON, R. R.: Ash Weight of Human Skeletons in Per Cent of Their Dry, Fat-Free Weight. Anat. Rec., **123**: 341-358, 1955.
17. TROTTER, M.; BROMAN, G. E.; and PETERSON, R. R.: The Density of Humeri of American Whites and Negroes. The Leech, **28**: 139-143, 1958.
18. TROTTER, M.; BROMAN, G. E.; and PETERSON, R. R.: Densities of Bones of White and Negro Skeletons. Am. J. Phys. Anthrop., **17**: 19-26, 1959.

DOCUMENTATION

The skeleton numbers, ages, dry, fat-free weights (gm.), volumes (cc.) and densities $\left(\frac{\text{weight}}{\text{volume}}\right)$ of ten bone series in each of 20 skeletons in four six-race groups are available by ordering Document number 6103 from the American Documentation Institute, Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington 25, D.C., remitting $1.25 for photoprints or $1.25 for thirty-five millimeter microfilm. Advance payment is required. Checks or money orders should be made payable to: Chief, Photoduplication Service, Library of Congress.

Clinical and Electromyographic Evaluation of Patients with Anterior Transposition of Peroneal Tendons*

BY WILLIAM H. BLODGETT, M.D., AND S. J. HOUTZ, M.S., DETROIT, MICHIGAN

From the Detroit Orthopaedic Clinic, Detroit

Transposition of one or both peroneal tendons to the dorsum of the foot has become an accepted orthopaedic surgical procedure in an attempt to re-establish dynamic dorsiflexion of a drop-foot due to the residuals of poliomyelitis. The effectiveness of the operation has been attested to by the clinical studies of Peabody, Mayer, Kuhlmann and Bell, and Reidy, Broderick, and Barr.

At the Detroit Orthopaedic Clinic some of the patients on whom this procedure was performed demonstrated progressive loss of strength of the transplant, recurrent heel-cord contracture, persistent drop-foot gait, or both a contracture of the heel cord and a persistent drop-foot gait even though good voluntary contraction of the transposed muscle was present initially. This led us to try to determine if the peroneal muscles, when placed in an antagonistic position, function automatically during dorsiflexion of the foot when walking, and if this change in automatic function has any influence on the results of the operative procedures.

Thus, the purpose of this study is twofold: to compare the electromyographic patterns during walking of the patients with anterior transpositions of the peroneal tendons with the patterns of normal subjects, and to correlate the recordings of muscle action potentials with clinical results.

APPARATUS

An eight-channel Offner, Type A, electroencephalograph adapted for electromyography was used for the recording of action potentials. It is a low-distortion machine which amplifies voltage from the contracting muscle fibers. Electrical activity was picked up with surface electrodes and recorded on an eight-pen, ink-writing dynograph with variable paper-speed controls. Prior to each experiment, all channels were adjusted to the same amplitude, calibrated by introducing a sixty-cycle current of known magnitude, and regulated to equalize the extent of the pen excursion.

The walking tread consisted of two plywood frames, ninety-six by ten inches, supporting a strip of galvanized iron, ninety-six by six inches, over which chicken wire was stretched tautly so that half an inch of separation was maintained between the wire and the iron. Plastic sheeting covered the wire to ensure comfort of the subject during barefoot walking. The weight of the body during the stance phase caused contact between the wire and galvanized iron, which closed a three-volt, dry-cell circuit. This activated a microswitch which deflected one of the writing pens of the dynograph. Thus, the swing and stance phases were indicated on the graph paper simultaneously with the muscle action potentials of the other seven leads.

METHODS

The subjects for this study were nine patients with anterior peroneal transplants after poliomyelitis and six normal young women and one man. Each of

* Aided in part by a grant from the Ford Foundation

TABLE I

PREOPERATIVE MUSCLE-STRENGTH EVALUATION*

Muscle	Patients Studied and the Ratings of Muscle Power								
	J.B.	L.E.	C.A.H.	C.H.	W.J.	M.O'R.	D.P.	K.S.	H.S.
Tibialis anterior	F	O	T −	O	T	O	T	O	P
Extensor hallucis longus	N	F	G −	F	F	F	F	G	G
Extensor digitorum longus	G	F +	G −	F	G	G	G	G	G
Tibialis posterior	F	F +	O	F	P	F	F	O	F
Gastrocnemius and soleus	G	N	N	F	P	F	G	G	N
Flexor digitorum longus†	N	F +	F +	G −	P	G	G	G	N
Flexor hallucis longus	N	T +	T +	G	T	F +	N	G	N
Peroneus longus	N	G	G	N	N	N	G	G	F +
Peroneus brevis	N	G	G	N	N	N	G	G	F +

*Muscle power was rated as normal (*N*), good (*G*), fair (*F*), poor (*P*), trace (*T*), and absent (O).

TABLE II

DATA ON PATIENTS WITH ANTERIOR TRANSPOSITION OF THE PERONEAL MUSCLE

Patient	Year and Month of Birth	Age* at Onset	Age* at Operation	Years between Onset and Operation
J.B.	June 1942	2	14	12
L.E.	June 1948	3	9	6
C.A.H.	April 1945	8	13	5
C.H.	January 1949	2	6	4
W.J.	June 1940	14	18	4
M.O'R.	September 1943	7	14	7
D.P.	May 1941	11	14	3
K.S.	December 1943	2	5	3
H.S.	October 1942	4	15	11

*Ages given in nearest years.

the normal subjects received a clinical evaluation of the feet; no abnormalities were found.

Originally, all forty-four patients at this Clinic on whom the operative procedure had been performed during the past five years were considered candidates for the study. Early in the investigation it became apparent that extensive paralysis or multiple operations influenced the electromyographic patterns of walking. Therefore, only patients with residual paralysis limited to the leg under study will be presented. In Table I and Table II are summarized the muscle strength and other pertinent information. All patients had received the usual physical therapy and protective bracing after the onset of the disease.

The decision to perform surgery was made at a preoperative consultation clinic in which four orthopaedists and two pediatricians participated. Except in the case of patient K.S., all operations were performed by the attending orthopaedic surgeons. In seven patients, just the peroneus longus tendon was moved; in the two other patients (C.A.H. and K.S.) both the longus and brevis were transposed. The tendons were implanted into the mid-tarsal bones in all but patients L.E. and H.S.; in these patients the tendon was sutured to the anterior tibial tendon near its insertion. In two patients (H.S. and M.O'R.) transposition of the extensor hallucis longus had been done several years prior to the peroneal transplant, but in both patients this procedure did not provide adequate dorsiflexion strength. The interim between the onset of disease and the surgical intervention ranged from three years to twelve years. The youngest child at the time of operation was five years, and the oldest was eighteen years. The average length of time that these patients had been under care at the Detroit Orthopaedic Clinic

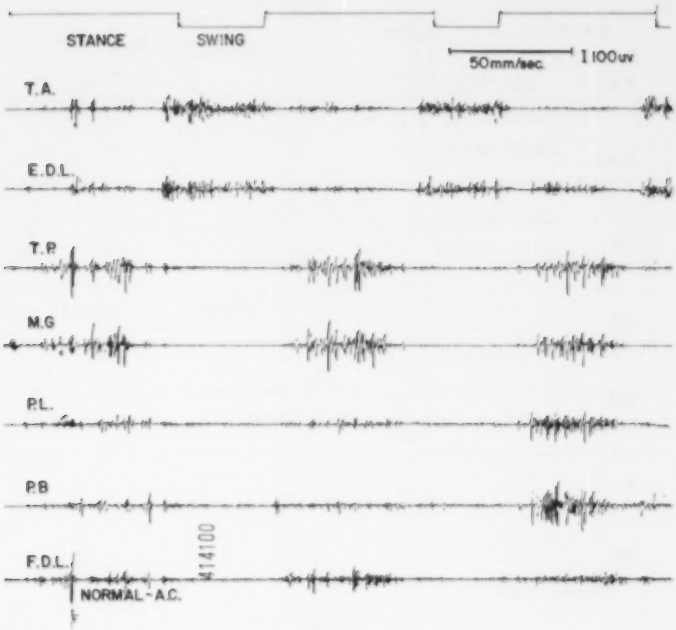


FIG. 1

Figs. 1 through 3: Electromyographic patterns of normal subjects during walking. The muscles recorded are: tibialis anterior (*T. A.*), extensor digitorum longus (*E. D. L.*), tibialis posterior (*T. P.*), medial gastrocnemius (*M. G.*), peroneus longus (*P. L.*), peroneus brevis (*P. B.*), and flexor digitorum longus (*F. D. L.*).

was six and one-quarter years; the longest and shortest periods were thirteen and three years, respectively.

In both the normal subject and the patients, electromyographic tracings of

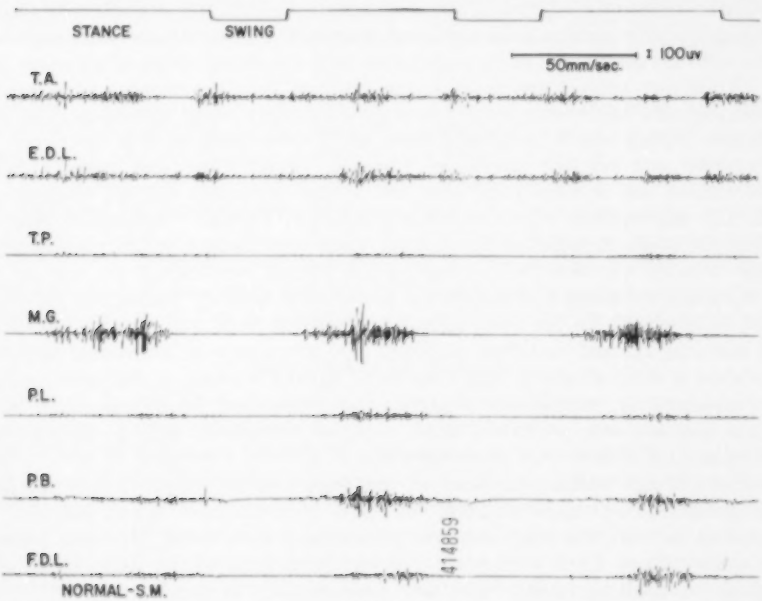


FIG. 2

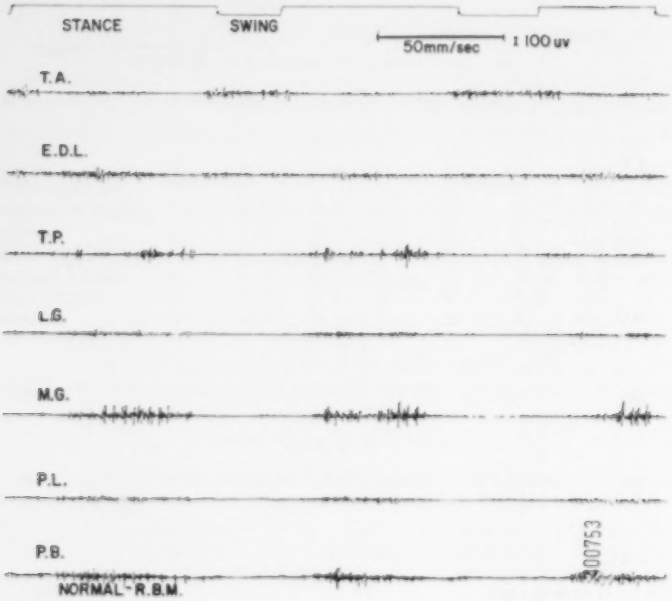


FIG. 3

the leg muscles were made during barefoot activities. The muscles sampled were the tibialis anterior, extensor digitorum longus, medial and lateral gastrocnemius, peroneus longus and brevis, tibialis posterior, and flexor digitorum longus. No attempt was made to record the electrical activity in patients who clinically demonstrated a completely paralyzed muscle. The action potentials were picked up with monel-metal surface electrodes, 0.8 centimeter in diameter, applied not less than two centimeters apart over the belly of the muscle in the direction of the fibers. Before the electrodes were attached, the skin was rubbed with electrocardiograph jelly until a mild erythema occurred. The electrodes were secured in place with an elastic collodion preparation and overlying strips of adhesive tape.

While each subject was in the supine position, electromyograms were recorded as the foot and ankle were moved through a complete range of motion. The other activities during which recordings were made were performed in the erect posture. Free and resisted inversion, eversion, dorsiflexion, and plantar flexion were carried out to be certain that the action potentials elicited corresponded with the appropriate observed muscle activity. Further verification that the action potentials recorded in the various leads were caused by the contraction of single muscles was obtained by having the subject circumduct the foot so that the variation in timing and magnitude of electrical activity during this movement could be observed. In the erect posture, recordings were made while the subject was standing relaxed and then walking. The myograms of additional activities, consisting in stair-climbing, deep knee-bending, and walking on the heels and toes, were obtained for subsequent analyses and evaluation. Each procedure in the upright position was performed after a period of relaxed sitting, during which time electrical silence was demonstrated.

The average walking distance on the ramp was fifty feet. Action potentials were recorded for approximately one-half the distance. Each subject was instructed to walk the nine-foot tread, turn, and walk back; this was repeated four or five times. Each person was indoctrinated prior to the study in an effort to obtain a walking routine that was characteristic of him.

The data obtained for analysis included the medical notes from a total of

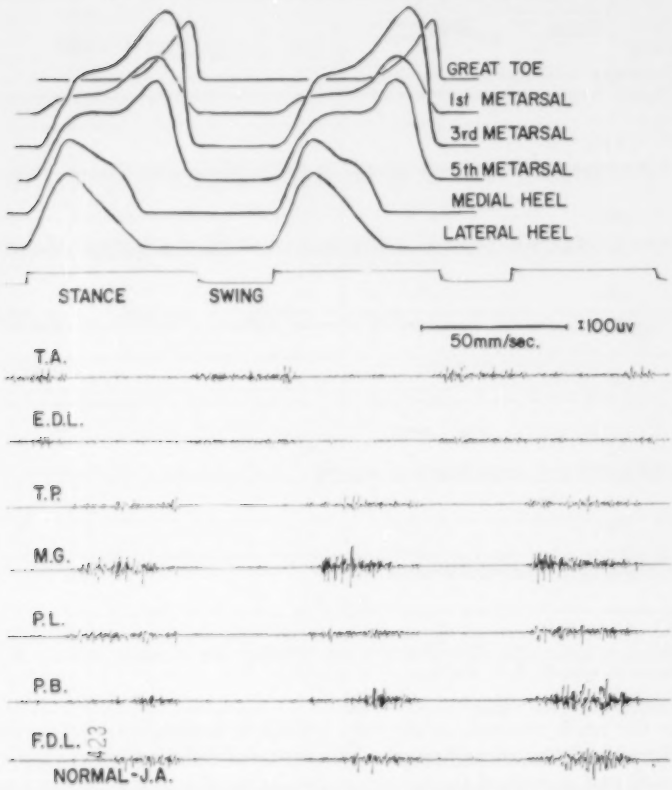


FIG. 4

The electromyographic pattern of the leg muscles of the normal subject during barefoot walking combined with the average weight-bearing points of pressure under the same circumstances (from Schwartz and Heath). The symbols for the muscles are the same as those in Fig. 1.

348 clinic visits that the nine patients made. This was an average of five visits per year, with the greatest number occurring during the year of the surgical procedure. The muscle evaluations and treatment given by the physical therapists were not included, but the results of the tests were recorded in the patients' charts and used for interpretative purposes. Fourteen electromyographic investigations were carried out on the patients. Each patient performed approximately thirteen different activities, making a total of 176 recordings. Every procedure was repeated four or five times, and the records for the prescribed activity were compared. An equal number of activities was performed by the normal subjects.

INTERPRETATION OF RESULTS

Figures 1, 2, and 3 are electromyographic patterns of three normal subjects during walking. Despite variations in gaits due to individual characteristics, the general timing of the phasic activity of each muscle was similar when compared among the subjects. The action potentials of the tibialis anterior and extensor digitorum longus occurred primarily during the swing phase, with varying degrees of contraction in the stance phase. From the other muscles sampled—the gastrocnemei, peronei, tibialis posterior, and long flexors to the toes—electrical activity was evoked during the stance phase, with periods of relative quiescence at the beginning and end of this movement. These results were not unlike those electromyographic patterns described by Sheffield, Gersten, and Mastellone and were

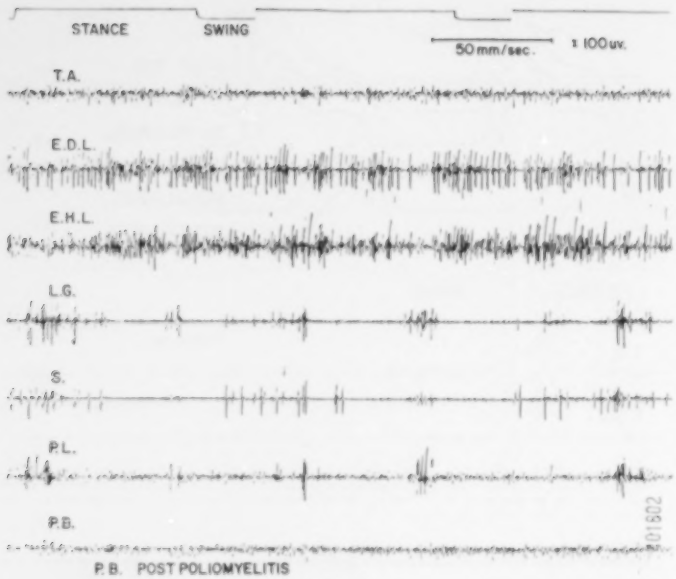


FIG. 5

Multilead electromyograms of the muscles of the right leg of a patient with residual paralysis due to poliomyelitis. This illustrates the complete loss of phasic activity. *E. H. L.* = extensor hallucis longus; *S* = soleus.

similar to the work reported in the very extensive investigation on fundamental studies of human locomotion from the University of California [15].

For a clearer picture of the biomechanics during the stance and swing phases, an electromyogram of a typical walking pattern was combined with the average weight-bearing pressures of specific areas of the foot as transmitted to the floor. Figure 4 is a diagram of sequential pressures exerted by the heel, metatarsal areas, and great toe, redrawn to appropriate scale from the published illustration by Schwartz and Heath. This shows that the dorsiflexors begin activity before the end of the stance phase and at the point of maximum pressure on the great toe mid-way in the double-weight-bearing period. They continue through the swing phase into the early portion of the stance phase as the fore part of the foot is lowered to the floor after the heel has begun weight-bearing. The remaining muscles contract after the heel strikes and at the point of maximum pressure on the heel. In general, their activity ceases as the heel is passively raised from the floor and the fore part of the foot is bearing maximum weight. In some instances, minimum contraction continues momentarily in the peronei and long toe flexors after activity in the gastrocnemei has terminated. In Figure 4 the duration of the swing phase is shown to be about one-half the stance phase.

Electromyograms of patients who have residual paralysis of the dorsiflexors due to poliomyelitis show a tendency toward an increase in the amplitude of the action potentials and a loss of the rhythmic muscle activity characteristic of the electromyograms of the normal subject (Fig. 5). This is particularly true of the long extensors of the toes and of the tibialis anterior, when present. Variability of pattern was seen also in the action-potential curves of the peroneus longus and brevis. The patterns of activity of the gastrocnemei deviate less frequently from the usual pattern than do those of the other muscles.

In one patient (W.J.), multilead electromyograms were obtained before operation and on three occasions after operation (Fig. 6). Progressive changes in the electrical-potential curves were demonstrated in these sequential recordings. In

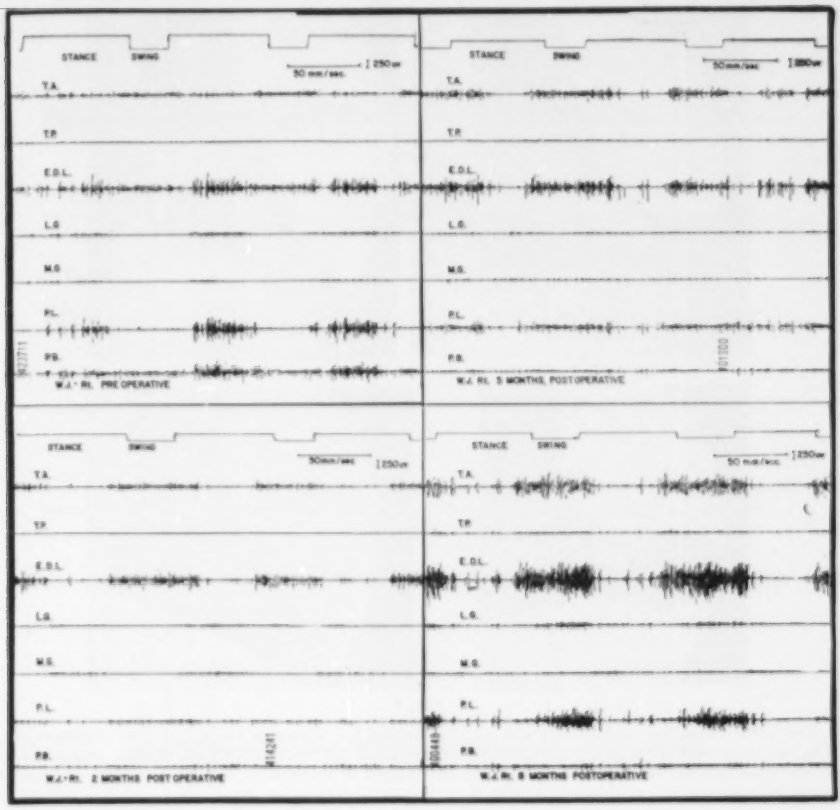


FIG. 6

One preoperative and three postoperative follow-up walking patterns of one subject (W. J.) with anterior transposition of the peroneus longus. In these records the rate of walking was the same. This illustration demonstrates a progressive shift of the peroneus longus activity to the swing phase in this muscle after transposition.

the walking pattern prior to surgery, the weak tibialis anterior and extensor digitorum longus showed continuous activity, although in the latter there appeared to be a greater amount of activity during the stance phase. The action potentials of the peroneus longus were normal, being present in the stance phase and entirely absent in the swing movement. Although the peroneus brevis had some phasic tendencies, it tended to be active throughout all phases of gait. There was negligible activity in the remaining muscles studied.

The electromyogram two months after operation showed that the peroneus longus was active weakly throughout both the swing and stance phases. The tibialis anterior and extensor digitorum longus began to demonstrate periods of activity interspersed with periods of quiescence. These bursts of activity occurred, in general, during the swing phase. This was not seen in the records prior to operative intervention. Two months after operation the patient was wearing a leg brace which was removed only for the duration of the study.

There was little change in activity of the dorsiflexors shown in the record five months after operation. The electrical activity of the peroneus longus appeared to have increased and to have developed some phasic characteristics. Just before this study was made the brace was removed permanently.

In the record eight months after operation the action potentials of the peroneus longus were grouped more in the swing phase. There was an increase in

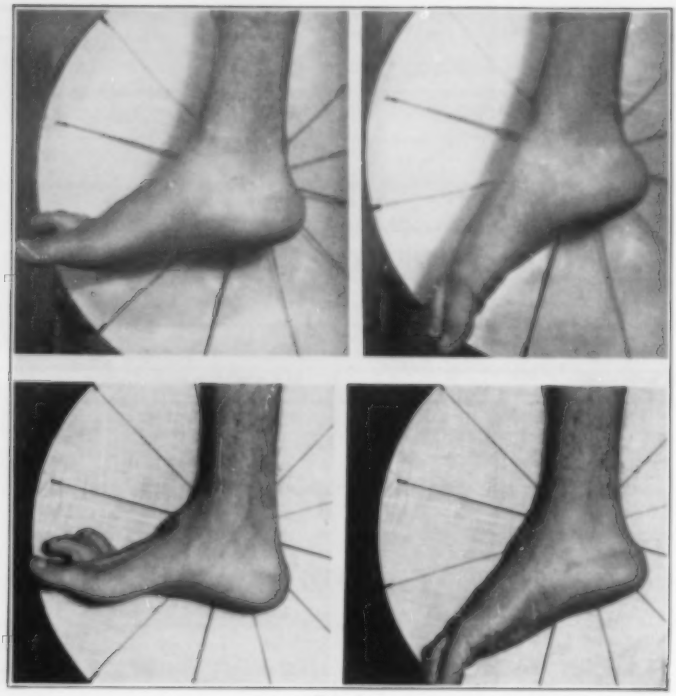


FIG. 7

Preoperative active dorsiflexion and plantar flexion are shown in the upper two photographs. The same movements after operation are shown in the lower illustrations for comparison. These photographs demonstrate the voluntary range of motion at the ankle of W. J., whose electromyograms are presented in Fig. 6.

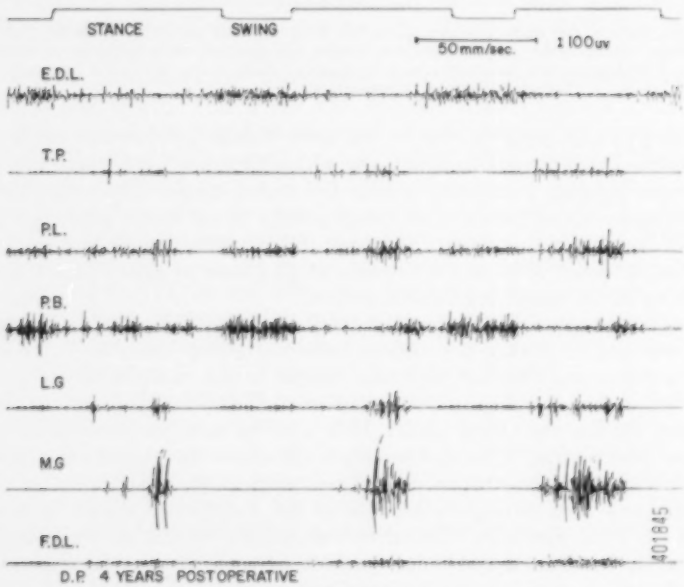


FIG. 8

Figs. 8 through 10: The walking patterns of three patients who demonstrated activity in the transposed peroneus longus during both the swing and stance phases.

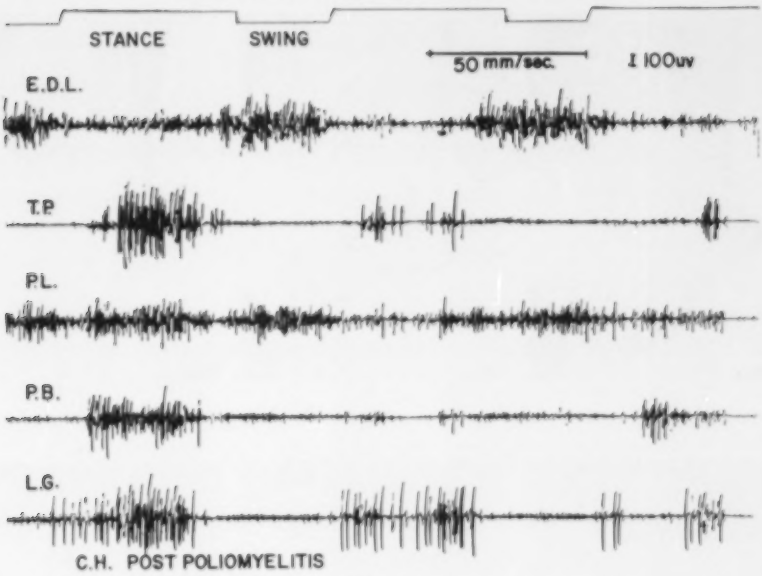


FIG. 9

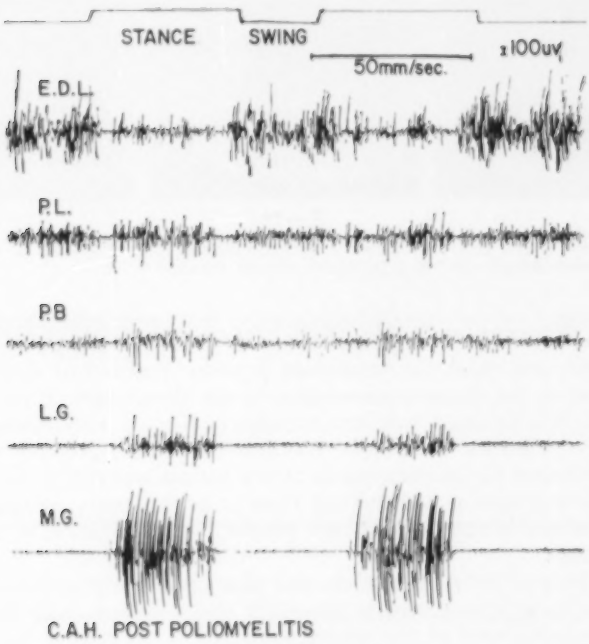


FIG. 10

amplitude of the action potentials, not only in this muscle, but also in the tibialis anterior and extensor digitorum longus. Postoperative photographs, made at this time, were compared with those made prior to surgery (Fig. 7). A slight increase in the patient's ability to voluntarily dorsiflex the foot was apparent.

When the records before surgery were compared with those of the final study, three changes were shown to have occurred: first, the tibialis anterior and extensor

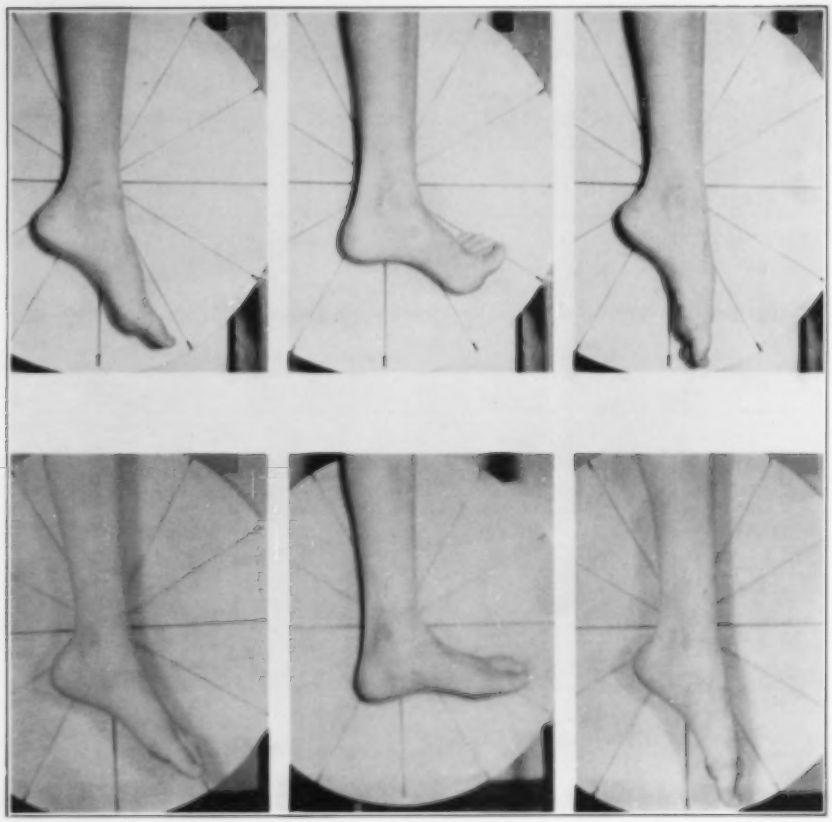


FIG. 11

Preoperative (upper) and postoperative (lower) photographs of C. A. H. with the foot relaxed, in active dorsiflexion, and in active plantar flexion.

digitorum longus had developed phasic activity corresponding to that seen in the normal person; second, the amplitude of the electrical potentials of these muscles had increased; and third, the transposed peroneus longus had shifted from its normal phase to the phase characteristic of the dorsiflexors. Prior to surgery, patient W.J. demonstrated only two muscles in the leg with clinically normal strength—the peroneals. These were considered to be the primary cause of the deformity. Whether the startling result in this patient was due to the elimination of the peroneus longus as a deforming force or to the active contraction of the transposed muscle is open to question; possibly, a combination of both factors was responsible.

On the basis of the studies before and after operation presented in Figure 6, the following criteria were used to assess the electromyograms of the remaining patients: the occurrence of the action potentials in relation to the swing and stance phases, the tendency toward rhythmicity of the action potentials, and the magnitude of electrical activity. The electromyograms were classed into three groups; those with phasic activity in both the stance and swing phases, those with continuous activity, and those with activity only in the stance phase.

The electromyograms of three patients showing action potentials during both phases of walking are illustrated in Figures 8, 9, and 10. The stance activity of the peroneus longus is retained, but there is a lull in the activity between the swing and stance phases. This we consider to be different from those postopera-

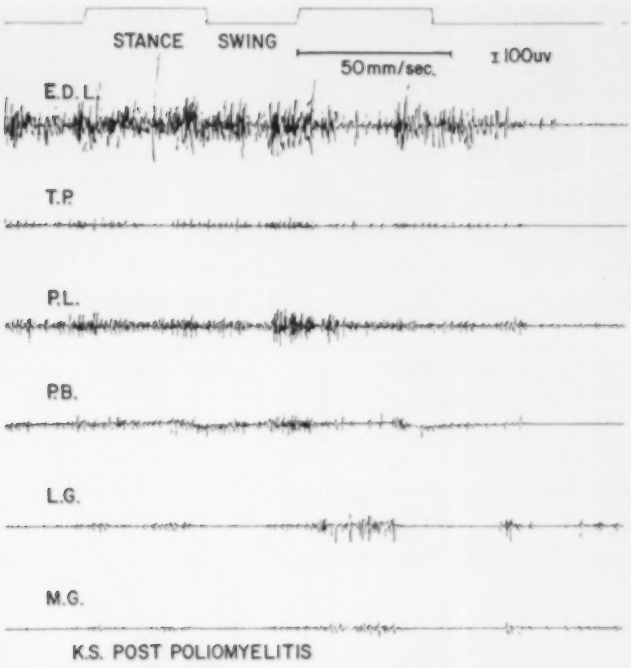


FIG. 12

Figs. 12 and 13: Postoperative gait patterns of two patients, demonstrating continuous action potentials in the peroneus longus.

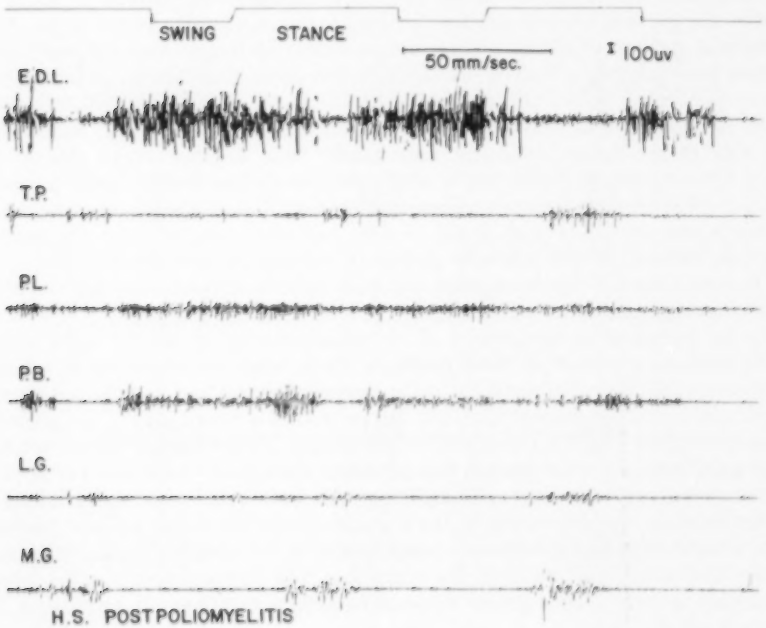


FIG. 13

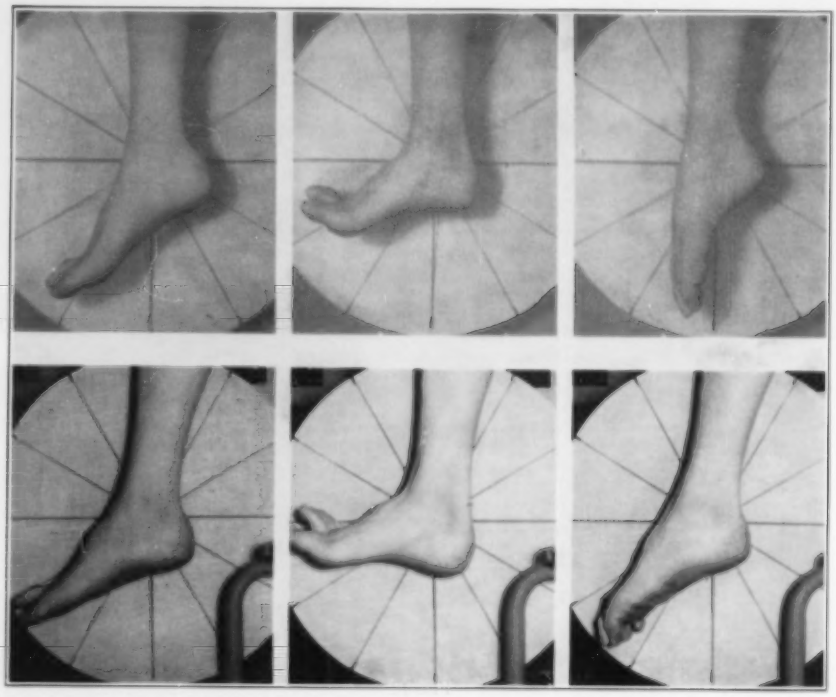


FIG. 14

A comparison of the voluntary range of motion of H. S. before (upper row) and after surgery. From left to right: resting foot, dorsiflexion, and plantar flexion.

tive records in which continuous activity of the muscle was present. In some instances, the peroneus brevis mimics the longus regardless of whether it has been transposed. Action potentials in the extensor digitorum longus were not continuous in these patients, being limited primarily to the same phase as the action potentials of this muscle in the normal person. In this group of three patients, the magnitude of the electrical activity shown in the electromyograms of D.P. approaches that seen in the normal subjects more nearly than do the magnitudes of the others. One patient (C.A.H.) had a triple arthrodesis two months prior to transposition of both peroneal muscles. How much this influences the electromyographic pattern is unknown. The photographs before and after surgery (Fig. 11) demonstrate an increase in this patient's ability to voluntarily dorsiflex the foot.

In two patients, the transposed peroneal muscle showed constant electrical activity throughout the swing and stance phases (Figs. 12 and 13). There is practically no variation in the pattern of electrical activity of the peroneus longus during walking in either of these patients. There were some periods of relative quiescence in the electrical activity of the extensors of the toes, but the occurrence of their active periods was inconsistent. In one patient's record (H.S.) the peroneus brevis contracted with the toe extensors and tended toward silence during activity of the gastrocnemius even though this apparent change of phase was not seen in the transposed peroneus longus. The photographs before and after operation (Fig. 14) demonstrate the differences in the amount of equinus in the relaxed position, in the amount of active dorsiflexion possible, and in the ability to actively plantar flex the foot which resulted from the operation. This limitation in plantar flexion (a decrease of nearly 30 degrees) appears to be due to a loss of mobility of the fore part of the foot.

The records of the remaining patients (Figs. 15, 16, and 17) show the elec-

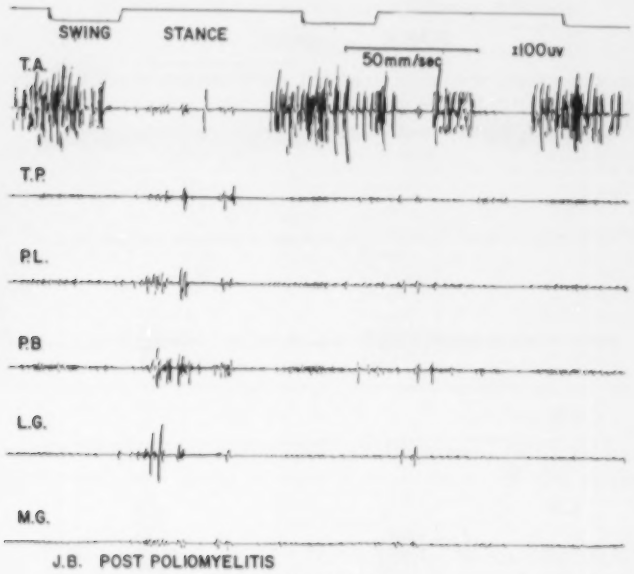


FIG. 15

Figs. 15 through 17: Electromyograms of three patients who did not demonstrate any change in the phasic activity of the transposed peroneus longus.

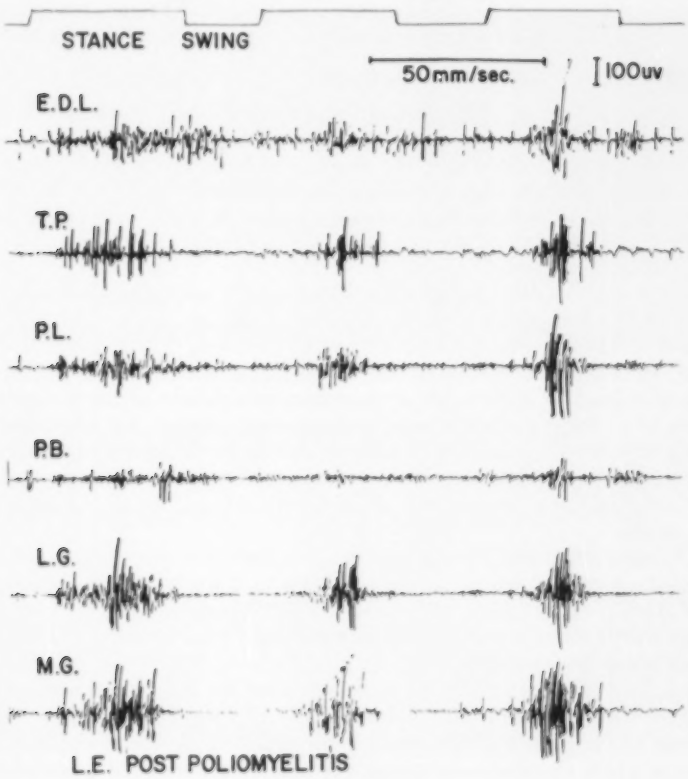


FIG. 16

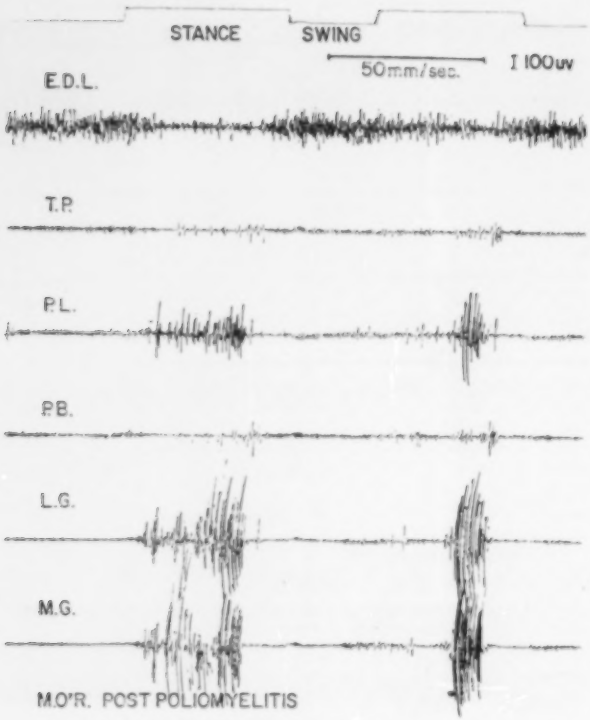


FIG. 17

trical activity of the peroneus longus to be predominant in the stance phase with insignificant activity in the swing phase. There is some fluctuation in the amplitude and timing of the action potentials of the extensor digitorum longus which varies from stride to stride. One of these patients (M.O'R.) could perform good voluntary dorsiflexion, as illustrated by the photographs before and after operation (Fig. 18).

In Table III the summary of the clinical results is correlated with an interpretation of electromyographic activity of the transplant.

The clinical evaluation was graded by the following criteria: *excellent* = no drop-foot, voluntary contraction of the transplant, dorsiflexion against resistance, and normal-appearing gait; *good* = voluntary contraction of the transplant, no drop-foot, no walking limitation, and perhaps some valgus; *fair* = partial drop-foot, voluntary dorsiflexion short of neutral (less than 90 degrees of dorsiflexion), brace not necessary, and tendency to development of heel-cord contracture; *poor* = drop-foot, slight or no contraction of the transplant, and brace or further operation necessary.

The electromyograms were graded as: *excellent* = complete transition from stance to the swing phase accomplished; *good* = definite increase in electrical potential during swing phase with a decrease or break in activity between the swing and stance phases; *poor* = continuous swing-phase and stance-phase activity, or no swing-phase activity.

The failure of the transplanted muscle to function during automatic walking was entirely independent of the patient's ability to voluntarily contract the muscle or even to dorsiflex the foot against resistance using the transplant. In all instances in which the electromyographic evaluations were *good* or *excellent*, the clinical results were *excellent* or *good*, *plus*. There were three patients with *good*

TABLE III

RESULTS AFTER TRANSPOSITION OF THE PERONEUS MUSCLE TO THE DORSUM OF THE FOOT

Patient	Years after Operation	Electromyographic Results	Clinical Results
J.B.	3	Poor	Good
L.E.	1	Poor	Poor
C.A.H.	1½	Good	Excellent
C.H.	2½	Good	Excellent
W.J.	¾	Excellent	Good, plus
M.O'R.	1	Poor	Excellent
D.P.	4	Good	Excellent
K.S.	10	Poor	Poor
H.S.	1½	Poor	Good

clinical results whose electromyographic readings were rated as *poor*. The one *poor* clinical result was found to have a *poor* rating on the electromyogram.

DISCUSSION

The multilead electromyographic tracings of leg muscles during normal walking confirm the split-second timing of the actions of the individual muscles which has been demonstrated by other investigators [13,15]. It seems apparent that this pattern of activity must be on an automatic basis and not subject to voluntary control.

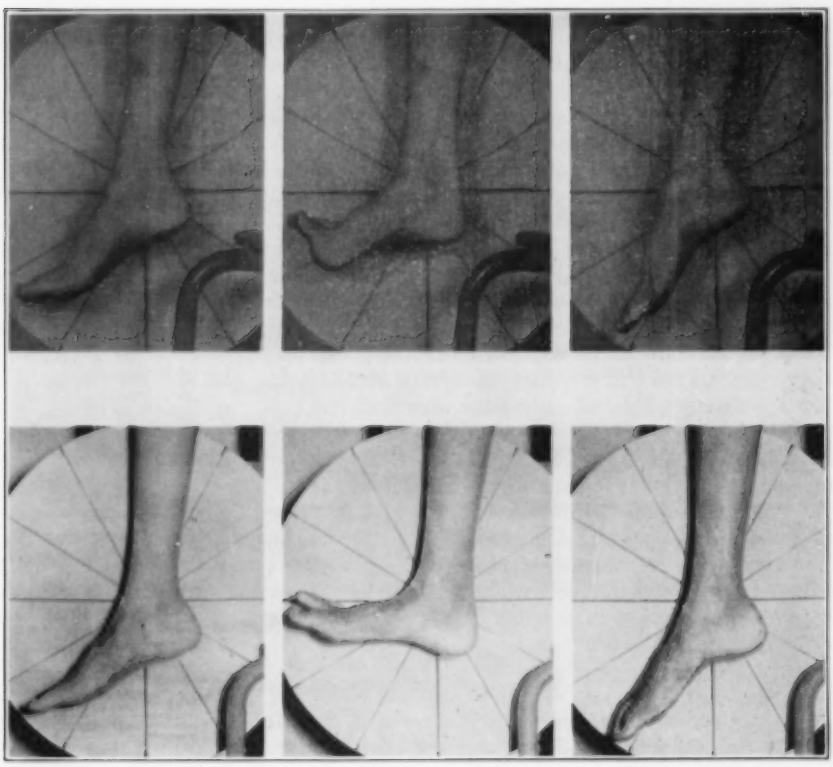


FIG. 18

Preoperative and postoperative photographs of M.O'R. From left to right: resting foot, dorsiflexion, and plantar flexion.

The specific phasic activity of individual muscles of the leg may be altered greatly by even a minor degree of muscle paralysis subsequent to poliomyelitis. These findings in the leg are in accord with the work of Weiss and Brown, who found that the coordination of the muscles of the thigh was lost after paralysis due to poliomyelitis.

The combined motions of the ankle and tarsal joints are responsible for the normal "universal-joint" movement of the foot in relation to the leg. Loss of contractile ability in any one of the muscles motivating these joints will change the dynamic function of the remaining active muscles. As Jones pointed out, except for the triceps surae, the muscles of the leg have multiple responsibilities in supporting and moving the components of the foot. When the foot is fixed on the floor, as in weight-bearing, muscle contraction results in changes in the position of the leg with reference to the floor rather than in changes in the position of the foot. A high degree of coordination of the actions of the various muscles is required. Disturbances in this coordinated function may be responsible for the extensive changes in phasic activity of leg muscles which occur in patients with residual paralysis.

The decision to perform anterior transposition of a peroneal tendon is usually based on the presence of weakness in dorsiflexion, adequate strength of the muscle to be transplanted and a dynamic valgus deformity. The muscle to be transposed is evaluated on the basis of its strength in voluntary movement of the free foot, as differentiated from the activity of the muscle while the foot is fixed on the floor during automatic walking. Frequently, at the time of transplant, this muscle has been functioning in a grossly distorted pattern for a long time. The time interval during which a muscle has lost its rhythmic activity may influence its ability to regain a rhythmical pattern of activity after transposition to a new location.

There is an abundance of reported material attesting to the clinical value of the procedure of anterior transposition of one or both peroneals, even though it was stated by Sperry, after a series of experiments in animal and man, that the capacity of the mammalian nerve system to readapt the function of its disarranged nerves and end-organs appeared to be quite limited. He said, in effect, that the transplanted muscle may work under voluntary control but could not be expected to work automatically.

The work of Gellhorn showed that proprioceptive reflexes in their phasic and static forms are an integral part of cortically induced movements. Therefore, after transposition of a tendon, the motion which activates the stretch reflex is modified and the proprioceptive stimulus is altered. This may account for the fact that in some patients, automatic contraction of the peroneus longus occurred during the swing phase. Close, in reporting his recent investigation, also described some patients who were able to accomplish this transition.

Good clinical results were obtained even when phasic shift in the transplant did not occur. This may result from the elimination of a dynamic valgus factor or because of the tenodesis effect of the transplant.

The limited number of patients presented in this study prevents the drawing of conclusions regarding the success or failure of this surgical procedure. The fact that even one transposed peroneus longus muscle can change phase should stimulate a search for factors which might influence such a transition of automatic activity. It may then be possible to predict those circumstances in which success or failure may be anticipated.

SUMMARY AND CONCLUSIONS

Multilead electromyographic studies and clinical evaluations were carried out on the leg muscles of nine children on whom anterior transposition of the peroneus longus had been done eight and one-half months to ten years previously. In one patient, one preoperative and three postoperative electromyographic studies were performed. The action-potential patterns of the leg muscles of seven normal

young adults were used as controls to aid in the interpretation of the pathological findings. The muscles studied were the tibialis anterior, extensor digitorum longus, tibialis posterior, peroneus longus, peroneus brevis, gastrocnemius, and flexor digitorum longus. The activities performed were simple range of motion and barefoot walking. The clinical evaluations of the end results were tabulated and correlated with the electromyographic analyses of the gait patterns.

The data presented support the following conclusions:

Normal automatic walking is made possible by the activity of the leg muscles which function with split-second timing in relation to the swing and stance phases and the double-weight-bearing portion of the stance phase.

In the normal walking patterns, the timing and phasic activity of these muscles generally have similar characteristics. There are certain variations among individuals, especially in the anterior tibial and toe extensors. The pattern of activity of the gastrocnemius shows the least individual variation in normal subjects.

The normal phasic activity of the muscles of the leg is frequently lost when there are various degrees of paralysis due to poliomyelitis.

After anterior transposition of the peroneus longus, transfer of activity of this muscle from the usual stance phase to the swing phase may occur. In one patient, a progressive and complete transition of activity to the swing phase was demonstrated by a series of electromyograms over a period of eight and one-half months.

In the small series studied good clinical results were obtained in all patients whose electromyograms demonstrated partial or complete shift of the activity of the transposed muscle to the swing phase of walking, but good clinical results were also obtained when electromyographic evidence of dorsiflexion activity was not present during the swing phase.

All patients with poor clinical results also had poor electromyographic evidence of transplant activity in the swing phase of walking.

Further studies should be carried out concerning the mechanism by which the transfer of phase occurs in the peroneus longus muscle.

REFERENCES

1. Adrian, E. D., and Bronk, D. W.: Discharge of Impulses in Motor Nerve Fibers. J. Physiol., **67**: 119-151, 1929.
2. Close, J. R., and Todd, F. N.: The Phasic Activity of the Muscles of the Lower Extremity and the Effect of Tendon Transfer. J. Bone and Joint Surg., **41-A**: 189-208, March 1959.
3. Gellhorn, Ernst: Physiological Foundations of Neurology and Psychiatry. Minneapolis, Minnesota, University of Minnesota Press, 1953.
4. Inman, V. T.; Ralston, H. J.; Saunders, J. B. deC. M.; Feinstein, Bertram; and Wright, E. W., Jr.: Relation of Human Electromyogram to Muscular Tension. Electroencephalog. and Clin. Neurophysiol., **4**: 187-194, 1952.
5. Jones, F. W.: Structure and Function as Seen in the Foot. Ed. 2. London, Baillière, Tindall and Cox, 1950.
6. Kuhlmann, R. F., and Bell, J. F.: A Clinical Evaluation of Tendon Transplantations for Poliomyelitis Affecting the Lower Extremities. J. Bone and Joint Surg., **34-A**: 915-926, Oct. 1952.
7. Licht, S. H., Editor: Some Clinical Applications of Electroneurophysiology, Especially Electrodiagnosis and Electromyography. New Haven, Connecticut, Elizabeth Licht, 1956.
8. Marinacci, A. A.: Clinical Electromyography. Los Angeles, San Lucas Press, 1955.
9. Mayer, Leo: Tendon Transplantations on the Lower Extremity. *In* Instructional Course Lectures, The American Academy of Orthopaedic Surgeons, Vol. 6, 1949, pp. 189-200. Ann Arbor, J. W. Edwards, 1949.
10. Peabody, C. W.: Tendon Transposition. An End-Result Study. J. Bone and Joint Surg., **20**: 193-205, Jan. 1938.
11. Reidy, J. A.; Broderick, T. F., Jr.; and Barr, J. S.: Tendon Transplantations in the Lower Extremity. J. Bone and Joint Surg., **34-A**: 900-908, Oct. 1952.
12. Schwartz, R. P., and Heath, A. L.: The Definition of Human Locomotion on Basis of Measurement. J. Bone and Joint Surg., **29**: 203-214, Jan. 1947.
13. Sheffield, F. J.; Gersten, J. W.; and Mastellone, A. F.: Electromyographic Study of the Muscles of the Foot in Normal Walking. Am. J. Phys. Med., **35**: 223-236, 1956.
14. Sperry, R. W.: The Problem of Central Nervous Reorganization after Nerve Regeneration and Muscle Transposition. Quart. Rev. Biol., **20**: 311-369, 1945.
15. Subcontractor's Report on Fundamental Studies of Human Locomotion and Other In-

formation Relating to the Design of Artificial Limbs Covering the Period from September 1945 through June 1947. Prosthetic Devices Research Project, Vol. 1. University of California, Berkeley, California, College of Engineering, Berkeley, 1947.

16. Weiss, Paul, and Brown, P. F.: Electromyographic Studies on Recoördination of Leg Movements in Poliomyelitis Patients with Transposed Tendons. Proc. Soc. Exper. Biol. and Med., **48**: 284-287, 1941.

DISCUSSION

The Effects of Continuous Compression on Living Articular Cartilage

(*Continued from page 49*)

My attention to pressure effects on cartilage was stimulated by the findings of cysts of the acetabulum at a point of great physical pressure. The movement of the femoral head distributes the force over that structure, but the maximum point of pressure of the acetabulum remains constant. When observing the hip joints, I was impressed by the fact that pressures less than those of catastrophic force just as surely destroyed the comfort and function of a joint, especially the weight-bearing joints. Pressure on the joint cartilages is gravitational as well as being due to muscular forces.

My approach to the subject, as a result of fortuitous findings in immobilized rat knees, demonstrated that the process of pressure destruction is gradual but persistently disastrous. The cartilage to which the fibrous tissue adhered was permanently damaged with little subchondral reaction. In the pressure areas the cartilaginous changes of tangential fibrillation and vertical invasion were consistently evident in rat knees immobilized for forty-five days or more. Not only were the cartilaginous changes evident, but subchondral fibrosis and cystic formation were constantly present. True changes in the joints can only be significantly understood when serial sections of the joint are made and compared with corresponding serial sections of the normal control of the same animal.

The subchondral changes impressed us as being irreversible, as Dr. Salter and Dr. Field also stated. It is important to note when there is damage to joint cartilages and subchondral areas that healing does not occur by osseous formation but by fibrous repair, and the cysts remain. As these insidious processes progress, ultimate destruction of a joint will follow as tragically as in the joints destroyed by the great forces, as exhibited by the authors.

Clinically, it is an important fact that these cartilaginous and subchondral lesions, being insidious in nature, are free from pain for long periods of time prior to the appearance of physical symptoms. This long interval between damage and pain in a patient makes me believe that if a condition is recognized early an improved circulatory status of the subchondral area may be created; thus, the joint is relieved of pain, and the damaged area is restored to reasonably good function.

Dr. David S. Grice, Philadelphia, Pennsylvania: Dr. Salter and Dr. Field demonstrated that continuous compression and immobilization cause necrosis of articular cartilage at the points of pressure. This has also been observed in the experiments of Evans and Eggers. Although the studies of Salter and Field record only that such necrosis occurs under continuous pressure, they still represent a significant contribution to our understanding of the behavior of hyaline cartilage. More studies are necessary to reveal the mechanism of this chondrolysis. It might be well to review some of the factors contributing to this necrosis.

Dr. Gardner recently reviewed the physiology of movable joints and pointed out that the weight-bearing in a joint conforms to the established principle of fluid-film lubrication. The weight thrust is not borne directly upon cartilage but is transmitted through a thin film of synovial fluid. The joint surfaces are incongruent during motion and are in contact only over a small area. In this respect they bear a close resemblance to the contact of a ball-bearing to a rotating shaft. In 1886, Reynolds showed that if two surfaces are inclined toward each other with a lubricant interposed, a wedge-shaped film is formed. The fluid enters at the base of the wedge and leaves at the apex. A pressure gradient develops, and the highest pressure is formed at the apex. This film possesses counterthrust sufficient to support a tremendous

(*Continued on page 90*)

The Ionization of Metal Implants in Living Tissues*

BY ALBERT B. FERGUSON, JR., M.D., PATRICK G. LAING, M.B., F.R.C.S., AND EDWIN S. HODGE, PH.D., PITTSBURGH, PENNSYLVANIA

From the Department of Orthopaedic Surgery, University of Pittsburgh, and the Physico-Chemical Section, Mellon Institute, Pittsburgh

The purpose of this paper is to report experimental evidence which indicates that ionization occurs around all types of metallic alloys currently in use in human beings regardless of how resistant to corrosion these alloys are thought to be. In the experiments to be reported here the concentration of metallic elements in the tissues around implants in rabbits has been determined with sufficient accuracy and in a sufficient number of determinations to permit valid statistical comparisons of the concentrations of metal around the various implants investigated. It should be emphasized, however, that this study concerns but one phase in the total picture of how a metallic alloy behaves in a living-tissue environment. The results reported here are the amounts of the metallic elements in the tissue around the implants at the time of sampling. They do not represent the total amount lost from any given specimen.

Some of the elements found in these experiments are not present in significant amounts in the tissues under normal conditions, whereas others, such as iron, are. It is assumed that the elements not ordinarily present in appreciable amounts are removed or excreted in some fashion and that those which are normally present presumably exist in some form of local equilibrium. The finding of these metals in appreciable quantities in the tissues around implants should clarify our thinking about the inertness of metal and have some bearing on the question of whether or not metal should be removed from the human body once it has served its purpose in therapy.

BACKGROUND

There are indications that the effects of metals on the tissues may become of increasing importance. Metallic implants have now been in use long enough for an appreciable number of patients to have had metal of various sorts embedded in their tissues for as long as thirty years. In our experience there have been a number of instances where sudden acute inflammation without evidence of bacterial infection has developed in the tissues after years of apparent quiescence.

There is also some evidence that certain metallic elements are in some way related to malignant disease. McDougall reported a bone sarcoma arising around a screw-and-plate device of dissimilar steels which had been in place for thirty years. Tietz, Hirsch, and Neyman reported a higher concentration of trace elements in cancerous tissue as compared with non-cancerous tissue. Schinz and Uehlinger produced local tumors in rats by intrafemoral transplants of chromium, and nickel has been implicated as a carcinogen by Hueper. Heath produced tumors in muscles with cobalt. The production of bone sarcomata by beryllium is well known [3], and copper produced teratoid tumors in roosters [1]. These findings should not, however, be interpreted to indicate that the use of metals should be abandoned for fear of tumor pro-

* This research was supported by United States Public Health Service Grant A1625.

TABLE I*

		With or Without Zeros	No. of Specimens	Parts per Million (*Means*)	Standard Deviation
Nickel	Controls	Without zeros	110	9.6	8.8
		With 22 zeros	132	8.0	9.3
	AISI 316	– –	66	40.5	41.2
	A-286	– –	16	54.9	20.2
	17.7 P.H.	– –	22	15.2	11.5
	Inconel X	– –	14	41.5	19.4
	Incoloy	– –	19	185.2	192.2
	Ni-onel	– –	20	31.3	11.4
	Inconel	– –	18	16.5	6.7
	Elgiloy	– –	10	46.8	52.3
	Hastelloy C	– –	18	15.7	8.2
Chromium	Controls	Without zeros	32	8.8	3.5
		With 43 zeros	75	3.7	4.9
	Co-Cr-Mo	– –	37	67.0	43.6
	AISI 316	– –	63	76.0	55.7
	Co-Cr-Ni-W	– –	9	74.9	99.8
	A-286	– –	16	26.9	24.8
	17.7 P.H.	Without zeros	12	15.2	14.3
		With 3 zeros	15	12.2	14.2
	Incoloy	– –	12	106.7	88.9
	Inconel	Without zeros	15	5.0	3.2
		With 1 zero	16	4.7	3.3
	Ni-onel	– –	20	16.7	10.2
	Inconel X	– –	14	7.6	1.6
	Elgiloy	– –	10	28.3	30.5
	Hastelloy C	– –	18	7.3	3.8
Molybdenum	Controls	Without zeros	40	5.8	3.3
		With 84 zeros	124	1.8	3.2
	AISI 316	– –	65	8.4	9.2

	Co-Cr-Mo	– –	46	11.5	8.9
	A-286	Without zeros	10	4.9	2.7
		With 9 zeros	19	2.6	3.2
	Ni-onel	Without zeros	12	3.5	0.8
		With 4 zeros	16	2.6	1.7
	Elgiloy	– –	10	7.9	5.0
	Hastelloy C	With 2 zeros	18	3.2	1.8
Titanium	Controls	Without zeros	193	13.3	18.2
		With 32 zeros	225	11.4	17.4
	Ti-implants	– –	25	236.8	168.4
	A-286	– –	17	27.1	32.4
Cobalt	Controls	Without zeros	71	11.3	8.1
		With 209 zeros	280	2.9	6.4
	Co-Cr-Mo	– –	54	76.7	45.9
	Co-Cr-Ni-W	– –	9	68.8	77.8
	Elgiloy	With 1 zero	10	33.5	29.4
	Hastelloy C	With 10 zeros	18	4.7	7.6
Zirconium	Controls	With 102 zeros	102	0.0	0.0
	Zr implants	– –	17	22.4	26.5
	Zr strips	With 2 zeros	10	98.7	98.7
Iron	Controls	– –	97	37.9	34.6
	AISI 316	– –	61	76.8	45.7
	A-286	– –	16	46.1	19.0
	Incoloy	– –	17	99.8	99.7
	Inconel	– –	18	26.0	13.6
	Inconel X	– –	14	39.3	18.0
	Ni-onel	– –	12	33.2	12.0
	17.7 P.H.	– –	28	34.4	15.4
	Elgiloy	– –	10	38.3	31.8
	Hastelloy C	– –	18	17.2	6.6

* Explanation for Table I will be found at the top of page 80.

LEGEND FOR TABLE I

This table records the data as mean concentrations of the elements found in the controls (normal tissue) and around the various implants together with the standard deviation. The data are grouped according to the element for which the tissues were analyzed. Since the method was not accurate to less than two parts per million, samples with this amount or less were recorded as zero. In instances where the control or tissue around an implant contained less than two parts per million of the element in question the data were analyzed first excluding and then including the samples with no measurable element. This is indicated in the column headed "With or Without Zeros". "Without Zeros" indicates that the statistical analysis was carried out not including the samples which contained "no" element; "With Zeros" indicates that all the samples were included in the analysis. Two dashes in this column indicate that more than two parts per million of the element were found in all the samples analyzed. The reason for analyzing the data both ways is to point out the differences in the results caused by including the specimens in the statistical analysis which contained no measurable element.

The terms and symbols used in this table are as follows:

AISI 316 is the stainless steel commonly used for surgical implants.

A 286 is a stainless steel of a different type which contains a small amount of titanium.

17.7 P.H. is precipitation-hardened stainless steel.

Inconel, incoloy, ni-onel, inconel X, elgiloy, and hastelloy C are the trade names of various alloys. Co-Cr-Mo is Vitallium, and Co-Cr-Ni-W is Haynes 25.

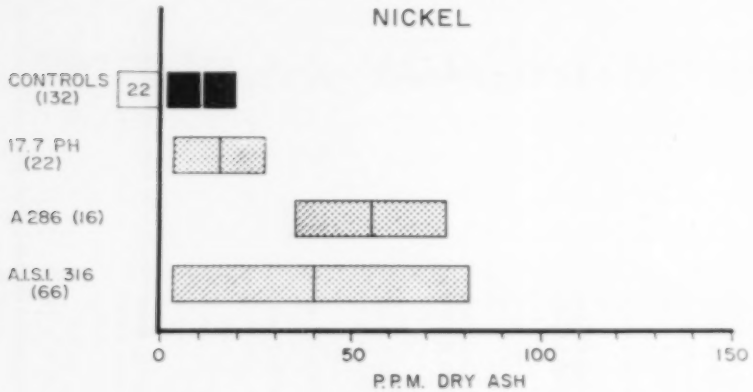


CHART I-A

Charts I-A through I-H: The various elements are contrasted with respect to individual elements. The results, in parts per million per dry ash, are shown as horizontal bars, the center line of which is the mean, with the length of the bar on each side of the line equivalent to one standard deviation. The number in parentheses below the name of the metal represents the number of specimens examined; the number in the flag on the upright represents the number of specimens in which the range of concentration of the metal was less than two parts per million. In the calculations these specimens were assumed to contain no metal.

duction. The common factor in all these experiments was that a long-term exposure was necessary before tumor formation occurred.

METHOD

In these experiments the embedded metallic implants were cylinders approximately one-eighth of an inch in diameter and one inch long and were finished as commonly supplied in manufactured form. The animals in which the implants were placed were albino rabbits weighing between 1,500 and 3,000 grams. They were kept on a standard laboratory diet of Rockland rabbit ration, carrots, and kale, and given tap water to drink. Spectrochemical analysis of the local water revealed a few trace metals in very small concentrations.

The metal cylinders were prepared for implantation by cleaning with a detergent, ether, and alcohol. They were then packed in paper envelopes and desiccated with phosphorus pentoxide and weighed. After being autoclaved they were inserted in the experimental animals. Only plastic instruments were

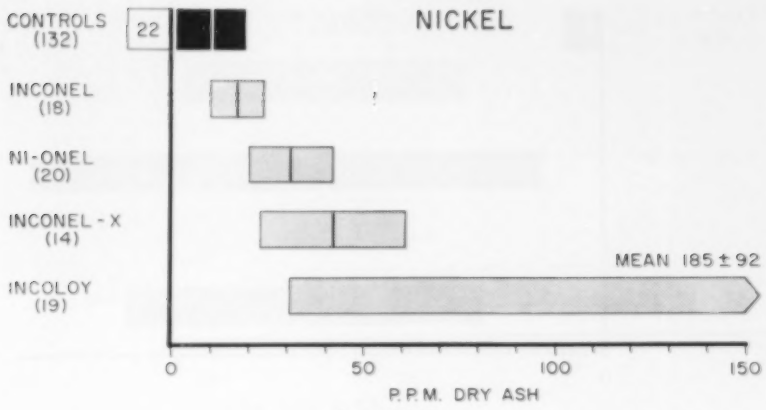


CHART I-B

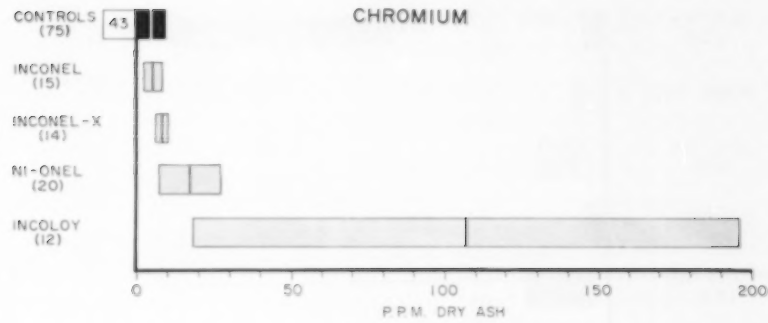


CHART I-C

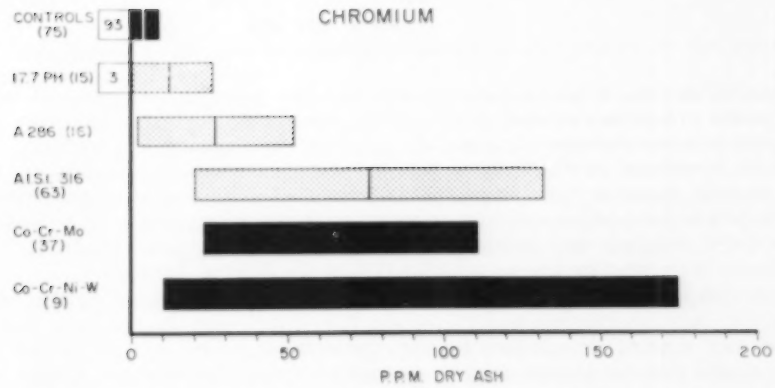


CHART I-D

used to avoid contamination of the implant and the tissues, except for metal knives which were used to make the pockets in the spinal musculature on either side of the vertebral column into which each individual cylinder was placed. It was usually possible to insert two or three cylinders of the same metal on each side. Dissimilar metals were never placed in the same animal.

When the cylinders were removed four to six months later, cube-shaped

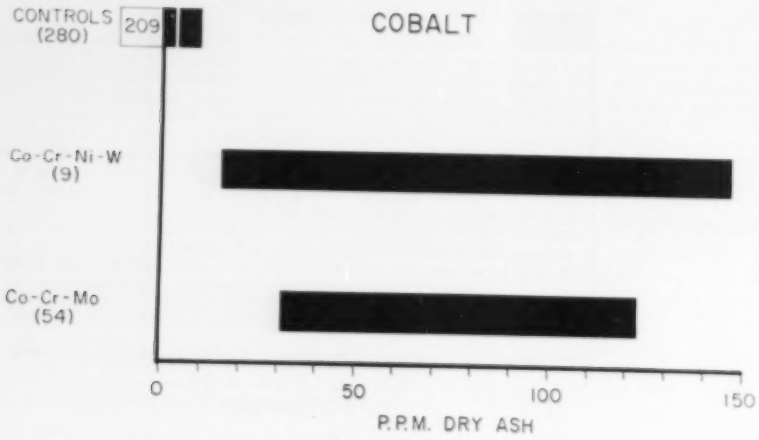


CHART I-E

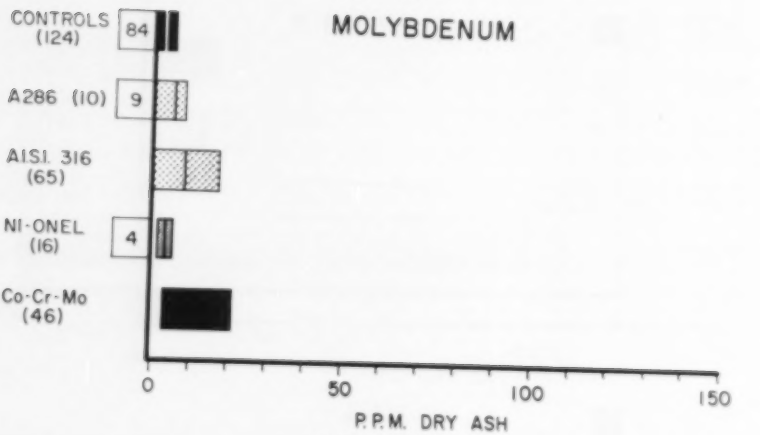


CHART I-F

blocks of muscle, weighing between one and two grams, were removed with the metal cylinders contained within them. Each metal cylinder was then removed, and the corresponding muscle specimen was pooled for analysis.

An occasional block of tissue was saved for histological examination. All the muscle implants were inspected and weighed. The pooled muscle specimens which were to be spectrochemically analyzed were placed in polyethylene bags, weighed, and quickly frozen for preservation until analysis. The analyses were done by one of us (E.S.H.) at the Mellon Institute.

In preparation for analysis, each sample was placed in an ignited and weighed platinum crucible and its weight was determined. It was then slowly dried and charred over a low Bunsen flame, after which it was burned off in a muffle furnace at 450 degrees centigrade. Ashing to constant weight required twenty-four hours. High-purity sulphuric acid was then added, after which the ash was dried, scraped with a platinum spatula, and ground in a boron-carbide mortar and pestle to a fine powder. The samples were then removed, weighed, and stored in 000 gelatin capsules. Equal weights of sample and high-purity graphite powder containing 0.01 per cent gallium oxide were put into a plastic vial and mixed by vigorous shaking in a dental amalgamator. Fifteen milligrams of each sample mixture was weighed and tamped into craters of special high-purity, one-eighth-inch electrodes for burning in

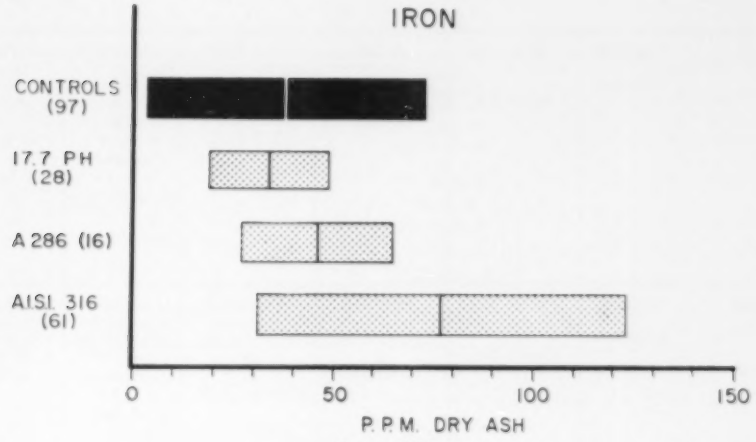


CHART I-G

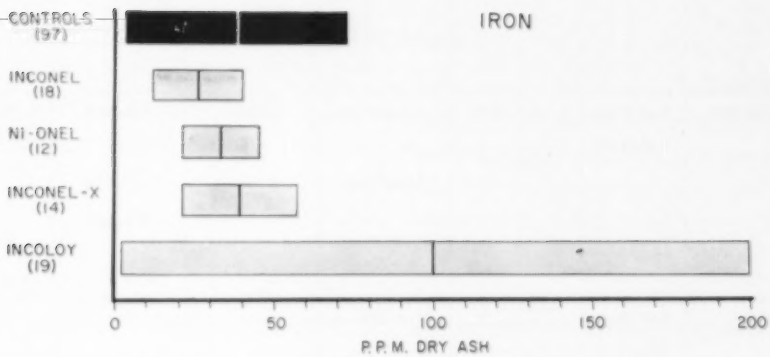


CHART I-H

the direct-current arc. A small hole drilled in the bottom of the crater gave more reproducible results.

A base containing sodium chloride, magnesium oxide, potassium metaphosphate, and calcium carbonate was prepared for use with a set of standards which contained all the test elements in concentrations ranging from 0.0001 per cent to 0.1 per cent in ten steps. Working curves were prepared over the whole range but were used principally in the range from 0.0001 to 0.01 per cent. The standards were mixed with graphite containing 0.01 per cent gallium oxide in the same fashion as the test samples. The standards and samples thus prepared were burned, and selected lines obtained in the spectrograms were read on a recording microphotometer. These data were used to prepare working curves and to make determinations of the quantities of the test elements present. The dry ash of each muscle sample constituted 0.9 per cent to 1.3 per cent of the weight of the wet muscle. The accuracy of the method could be reduced to within two parts per million.

RESULTS AROUND METAL IMPLANTS

The results, including the number of specimens, means, and standard deviation, are given in Table I. The amount of each metallic element occurring in muscle which had not been exposed to the implants was used as the control in each instance. The results for the different implants become mean-

TABLE II*

Nickel	Controls	AISI 316	A 286	17.7 P.H.	Inconel X	Incoloy
	Controls – – – – AISI 316	– – – – n A 286	– – + + + + + + 17.7 P.H.	– – – – n n – – – – Inconel X	– – – – – – – – – – – – – – – Incoloy	
Chromium	Controls	Co-Cr-Mo	AISI 316	Co-Cr-Ni-W	A 286	17.7 P.H.
	Controls – – – – Co-Cr-Mo	– – – – n AISI 316	– – – – n n Co-Cr-Ni-W	– – – – + + + + + + A 286	– – – + + + + + + + + + n 17.7 P.H	
Molybdenum	Controls	AISI 316	Vitallium	A 286	Ni-onel	Elgiloy
	Controls – – – – AISI 316	– – – n Vitallium	n + + + + + A 286	n + + + + n Ni-onel	– – – – n n – – – – – Elgiloy	
Titanium	Controls	Titanium	A 286			
	Controls – – – – Titanium	– – – + + + + A 286				
Cobalt	Controls	Vitallium	Haynes 25	Elgiloy		
	Controls – – – – Vitallium	– – – – n Haynes 25	– – – – + + n Elgiloy			
Zirconium	Controls	Zirconium Rods	Zirconium Strips			
	Controls – – – – Zirconium Rods	– – – – – – Zir. Strips				
Iron	Controls	AISI 316	A 286	Incoloy	Inconel	Inconel X
	Controls – – – – AISI 316	n + + A 286	– – – – n – Incoloy	n + + + + + + + + Inconel	n + + n + – Inconel X	

* An explanation for Table II will be found at the top of page 86.

TABLE II

Ni-onel	Inconel	Elgiloy	Hastelloy C		
− − − − n + + + + − − − − n + + + Ni-onel	− − − − + + + + + n + + + + + + + + + + + Inconel	− − − − n n n n + n − Elgiloy	− − − + + + + + n + + + + + + + + + + + n + Hastelloy C		
Incoloy	Inconel	Ni-onel	Inconel X	Elgiloy	Hastelloy C
− − − − − n n − − − − − Incoloy	n + + + + + + + + + + + + + + + + + + Inconel	− − − − + + + + + + + + + n n + + + + − − − − Ni-onel	− − + + + + + + + + + + + n + + + − − + + Inconel X	− − − − + + + n n n + − − n − Elgiloy	− − + + + + + + + + + + + + n + + + + − + + + n + + Hastelloy C
Hastelloy C					
n + + + + n n + + Hastelloy C					
Ni-onel	17.7 P.H.	Elgiloy	Hastelloy C		
n + + n + n n Ni-onel	n + + + + + + + n n n 17.7 P.H.	n + n n n n n n Elgiloy	+ Hastelloy C		

LEGEND FOR TABLE II

This is a series of tables, one for each element. Each table records the statistical significance of the differences in concentration of the element in question in the tissues around the implants which are listed at the top of the vertical columns and in the control tissues. These tables provide a simple way of visualizing whether the results obtained for each implant were significantly different from those obtained for the other implants with respect to a particular element.

The meanings of the symbols in the vertical columns are as follows:

n = equivalent; $P > 0.05$
+ or − = $0.05 > P > 0.01$ = possibly significant
++ or −− = $0.01 > P > 0.001$ = probably significant
+++ or −−−− = $0.001 > P > 0.0001$ = significant
++++ or −−−−− = $P > 0.0001$ = highly significant

To use the table, read from left to right and from below up. For example, the controls almost always contained less element than the tissues around the implants. Thus, for nickel, reading to the right from the word "controls" at the bottom of the column headed "Controls", it is seen that the concentration of nickel in the controls was highly significantly less (−−−−−) than in the tissues around the AISI 316 and A 286 implants and probably significantly less (−−) than that in the tissues about 17.7 P.H. steel, and so forth.

As another example the tissue concentration of nickel around 17.7 P.H. steel was highly significantly less (−−−−−) than that about inconel X and ni-onel and equivalent (n) to that around inconel and elgiloy as indicated by reading from left to right starting at "17.7 P.H." at the bottom of the column headed "17.7 P.H." However, to compare the nickel concentration around 17.7 P.H. steel with that around the implants to the left of 17.7 P.H. in the table (A 286 and AISI 316) and with that in the controls, it is necessary to read from below upward in the 17.7 P.H. column, reversing the signs. Thus, the tissue concentration of nickel around 17.7 precipitation-hardened steel was highly significantly less (++++ changed to −−−−) than that around A 286 steel, and so on. Again, the concentration around hastelloy C was possibly significantly less than that around elgiloy (+ changed to −), equivalent (n) to that around inconel X, and so forth, reading upward from the bottom of the last column headed "Hastelloy C".

---

ingful when contrasted with each other statistically, as has been done in Table II.

Nickel: The concentration of nickel around the 17.7 precipitation-hardened steel containing 6.5 to 7.5 per cent nickel and 70 per cent iron was similar to that around inconel which contains 78 per cent nickel and 7 per cent iron. Both these alloys were surrounded by less nickel than the other alloys with which they were contrasted. Incoloy, with a combined nickel-cobalt concentration of 30 to 34 per cent, gave a concentration of nickel in the surrounding tissues which was consistently larger than that which occurred around the other alloys. AISI 316 steel, with a concentration of 13.4 per cent nickel, was surrounded by more nickel than was 17.7 precipitation-hardened steel, in which the nickel concentration is only 6.5 to 7.5 per cent.

Chromium: Less chromium was found around inconel (14.3 per cent chromium) than around any of the other implants. Only around 17.7 precipitation-hardened steel (16.7 per cent chromium) did the concentration of this element even approach the low level around inconel. The tissue concentration of chromium around the cobalt-based alloys, Vitallium (Co-Cr-Mo) and Haynes 25 (Co-Cr-Ni-W), was not less than that around the other alloys to a highly significant degree. Vitallium (28.3 per cent chromium) and AISI 316 steel were quite similar, but both had a higher tissue concentration of chromium than had AISI 316 steel, hastelloy C, and elgiloy. Incoloy (19 per cent chromium) showed a significantly higher tissue concentration of chromium than all the other alloys with which it was compared.

Molybdenum: The differences in the concentration of this element in the tissues around the various implants were not so marked as the differences in concentration of nickel and chromium. There was no significant difference in the tissue concentrations of molybdenum around AISI 316 steel and Vitallium. A 286 steel (1.0 to 1.5 per cent molybdenum), hastelloy C (17.0 per cent), and ni-onel (2.9 per cent) were surrounded by significantly less molybdenum than were the other alloys.

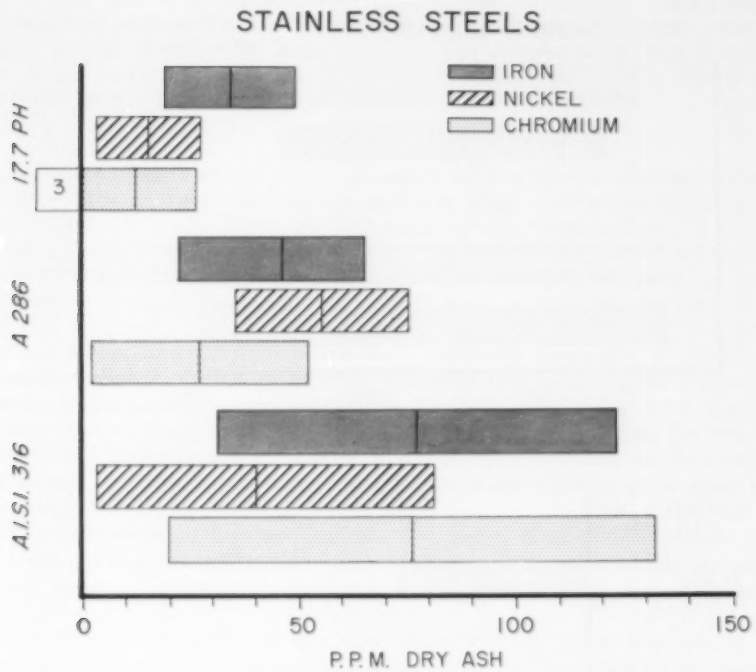


CHART II-A

Charts II-A through II-D: The results for several of the elements from one alloy are grouped together to emphasize the fact that if there is much ionization of one element in an alloy there tends to be much ionization of all the elements in the alloy.

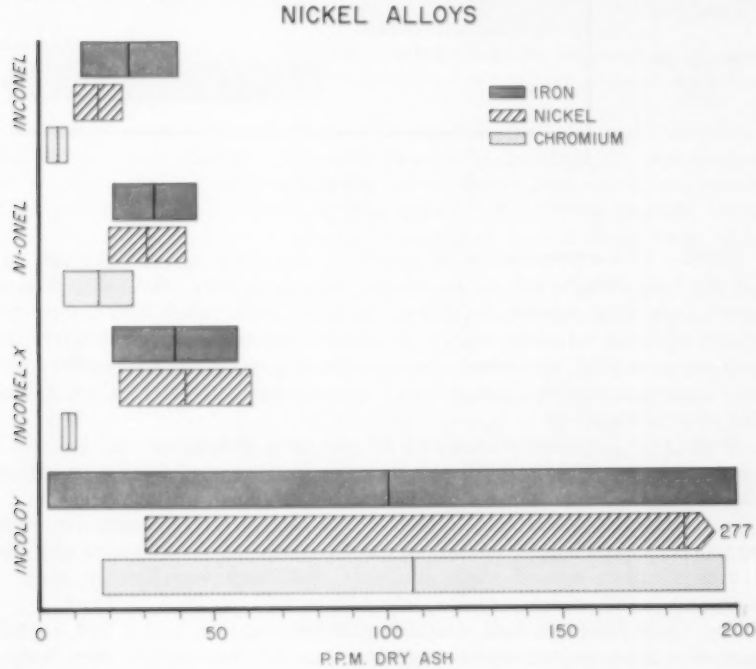


CHART II-B

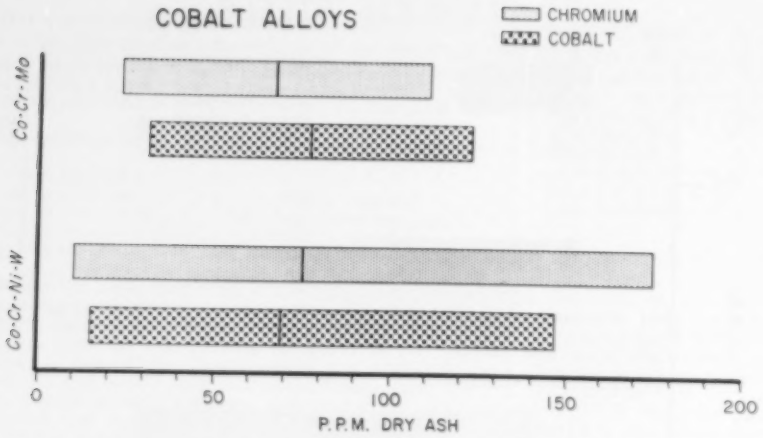


CHART II-C

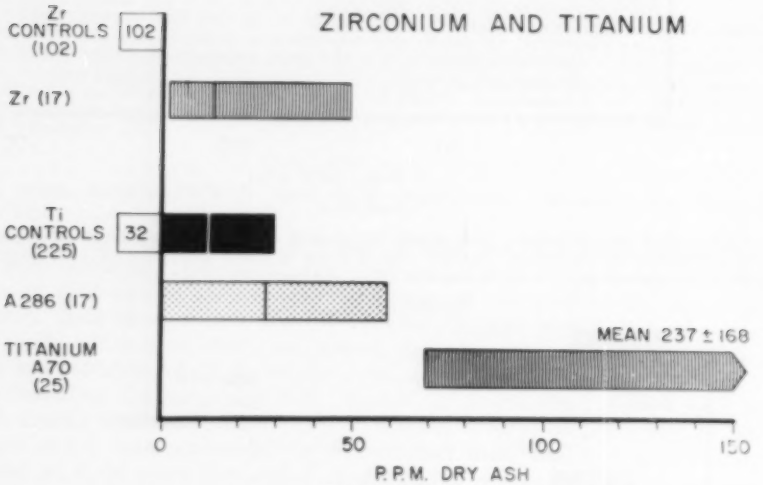


CHART II-D

Cobalt: The concentration of cobalt in the tissue around Haynes 25 (46 to 53 per cent cobalt and 14 to 16 per cent tungsten) was not significantly different from that around Vitallium (61.9 per cent cobalt and no tungsten). Implants of both of these alloys increased the cobalt concentration in the tissues to a highly significant degree above normal. Elgiloy (40 per cent cobalt) caused a slightly higher tissue concentration of cobalt than did either of the other two alloys.

Zirconium: Implants containing 97 per cent zirconium and 1.9 per cent hafnium caused a highly significant increase in the concentration of zirconium in the tissues.

Titanium: The titanium implants, containing 90 per cent or more of titanium, very significantly increased the titanium concentration in the tissues. The concentration around these implants was very significantly more than that around A 286, which contains only 1.75 to 2.25 per cent titanium.

Iron: Only AISI 316 steel, containing 64 per cent iron, and A 286, containing 48 per cent iron, caused tissue concentrations of iron which were very significantly higher than normal. The 17.7 precipitation-hardened steel, with 70

per cent iron, had a concentration of iron in the surrounding tissue which was equivalent to the iron concentration in normal tissue. Because iron is normally present in tissue in large amounts, the differences in iron concentration found around these implants are not so significant as the differences which were found in the tissue concentrations of the other metals.

DISCUSSION

The reproducibility of the results obtained by the spectrochemical method of analysis used in these experiments and the large number of highly significant differences in the tissue concentrations of the metallic elements found in these biological experiments give some credence to the results obtained.

As previously stressed, the results of these experiments are only the concentrations of the individual elements around each implant at the time that the animal was sacrificed. They give no indication as to whether the concentrations around the parent implants have increased, decreased, or remained constant since the time of implantation. However, only a negligible difference was found in the concentrations around the implants removed at four months when they were compared with those removed at six months. When the weight changes of the implants are correlated with the concentrations of the metals in the tissues and the duration of implantation, some light may be shed on this question.

The results reported here do establish the fact that metallic elements appear in the tissues around metal implants in concentrations which are significantly greater than normal, no matter how non-corrosive these implants have been found to be in other environments.

When it comes to selecting the ideal alloy for use as an implant, it is obvious that the selection cannot be based purely on the amount of metal which has been ionized and identified in the tissues, as was done in these experiments. The toxicity and other actions of these various metallic elements and their ions must be explored as well as the properties of the alloys themselves, which may be of significance. In many cases these questions await clarification.

The large concentration of titanium around titanium implants is of interest, as is the relatively small concentration of zirconium around zirconium implants.

Corrosion is a process not subject to general rules. It varies markedly with individual environments. Titanium, markedly resistant to corrosion in chloride solutions in industrial usage, has been shown here to be surrounded by a high concentration of titanium when implanted in living tissues. Living tissue is a dynamic, protein-containing, oxygenated environment from which nitrogen has been largely excluded.

Inconel, inconel X, 17.7 precipitation-hardened steel, A 286 steel, and hastelloy C were particularly outstanding in having small concentrations of metals around them. The tissue concentrations of metals around Vitallium, elgiloy, and AISI 316 steel were near the median. It is of interest that the concentration of the elements found in the tissues around these implants did not correlate with the concentration of the elements in the alloy, except in the case of titanium.

In general, it was observed that a metal alloy with a significantly high concentration of one element around it also tended to have high concentrations of others. This is brought out when the results are drawn up diagrammatically, as in Charts II-A through II-D. The results are more readily visualized by means of these figures, and, accordingly, will bear perusal when the attempt is made to contrast one metal with another.

REFERENCES

1. Falin, L. I., and Anissimova, V.: De la pathogénie des tumeurs expérimentales teratoïdes des glandes génitales: tumeur teratoïde de la glande séminale du coq causée par l'injection

d'une solution de sulfate de cuivre. Bull. Biol. et Méd. Expér. U.R.S.S., **9**: 518-520, 1940.
2. Heath, J. C.: Cobalt as Carcinogen. Nature, **173**: 822-823, 1954.
3. Hueper, W. C.: Experimental Studies in Metal Cancerigenesis. VI. Tissue Reactions in Rats and Rabbits after Parenteral Introduction of Suspensions of Arsenic, Beryllium, or Asbestos in Lanolin. J. Nat. Cancer Inst., **15**: 113-129, 1954.
4. Hueper, W. C.: Experimental Studies in Metal Cancerigenesis. IV. Cancer Produced by Parenterally Introduced Metallic Nickel. J. Nat. Cancer Inst., **16**: 55-73, 1955.
5. Hueper, W. C.: Experimental Studies in Metal Cancerigenesis. VII. Tissue Reactions to Parenterally Introduced Powdered Metallic Chromium and Chromite Ore. J. Nat. Cancer Inst., **16**: 447-469, 1955.
6. McDougall, A.: Malignant Tumour at Site of Bone Plating. J. Bone and Joint Surg., **38-B**: 709-713, Aug. 1956.
7. Schinz, H. R., and Uehlinger, E.: Der Metallkrebs. Ein Neues Prinzip der Krebserzeugung. Zeitschr. f. Krebsforsch., **52**: 425-437, 1942.
8. Tietz, N. W.; Hirsch, E. F., and Neyman, Benjamin: Spectrographic Study of Trace Elements in Cancerous and Noncancerous Human Tissues. J. Am. Med. Assn., **165**: 2187-2192, 1957.

DISCUSSION

The Effects of Continuous Compression on Living Articular Cartilage

(Continued from page 76)

load, even when the film is thin. MacConaill estimated the effective thickness of this film in joints as 50 micra. Fluid-film lubrication can survive a load of 900 pounds per square inch. Jones, in 1934, found that the coefficient of friction in an unlubricated joint is fourteen times that of a lubricated one. He also observed that if an unlubricated joint was worked for four hours, the cartilage would be worn down to bone.

What is it that provides this lubrication film? The viscosity of synovial fluid is related to the presence of hyaluronic acid. Anything that alters the concentration and the state of hyaluronic acid in the joint will affect the viscosity of the synovial fluid and the fluid-film lubrication. Recently, Jebens and Monk-Jones wrote that there is a decrease of viscosity of normal synovial fluid with age and acute trauma of a normal joint. It is tempting to speculate about the effect of weight-bearing on articular cartilage in joints with decreased viscosity of synovial fluid. How can this be measured? What is the relation of this to degenerative arthritis?

Another factor to be borne in mind in any problem involving necrosis of cartilage is the role of plasmin. Lack showed that plasmin, a proteolytic enzyme, will digest the mucoprotein of cartilage, causing proteolysis, and that plasmin may be activated in synovial fluid by trauma to synovial tissue and cartilage. It is also activated by bacteria. It, therefore, becomes apparent that with decreased viscosity in the change in the synovial-fluid hyaluronic acid and secondary trauma, the presence of plasmin may produce a situation in which there is decreased viscosity, greater pressure on the cartilage, and, perhaps, as Dr. Salter and Dr. Field have shown, destruction of cartilage may occur. All these factors may play a role in situations other than that of the tremendous compression force that produces necrosis of cartilage, as pointed out by Dr. Salter and Dr. Field.

The authors' contribution is very significant; they have shown that there is necrosis with compression. The interpretation of all the factors involved will require further experimentation; I hope they will continue in this productive study.

Heterogenous and Autogenous-Tendon Transplants

An Experimental Study of Preserved Bovine-Tendon Transplants in Dogs and Autogenous-Tendon Transplants in Dogs *†

BY J. EDWARD FLYNN, M.D., JOHN T. WILSON, M.D., CHARLES G. CHILD, III, M.D., AND JAMES H. GRAHAM, M.D., BOSTON, MASSACHUSETTS

From the Department of Surgery, Tufts University School of Medicine, and First (Tufts) Surgical Service, Boston City Hospital, Boston

This article reports a study of the gross and histological changes in and around heterogenous and autogenous-tendon transplants at varying periods after insertion of the transplant. Three different experiments were done, and the procedures were as follows: In Experiment 1, young bovine tendons preserved in homologous plasma were transplanted into surgically prepared defects in dogs' tendons. In Experiment 2, irradiated and frozen fetal bovine tendons were used, and in Experiment 3, autogenous-tendon transplants were used in dogs in a similar fashion.

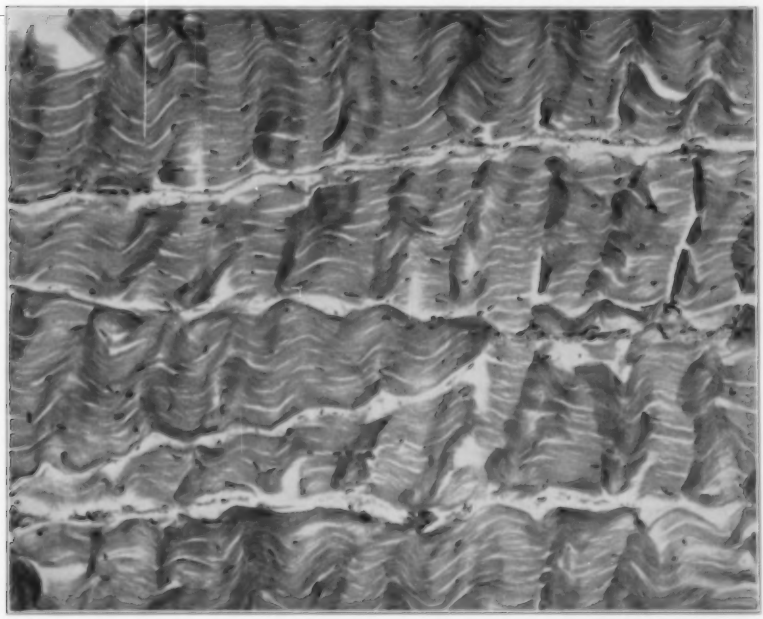


FIG. 1

Young bovine tendon (× 165). The fasciculi are separated by septa from the epitendineum and are composed of fibrils of collagen. The nuclei of the tendon cells (cells of Ranvier) are spherical, oval, and flat. The dog's tendon has a similar appearance.

* Presented at the Annual Meeting of the American Society for Surgery of the Hand, Chicago, Illinois, January 23, 1959.

† This project was supported by a grant from The Squibb Institute for Medical Research.

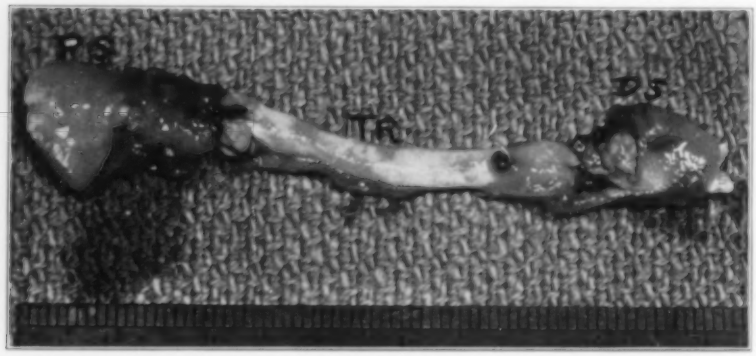


FIG. 2

Young bovine tendon preserved in homologous plasma. Gross specimen, one week after operation, shows *PS*, the proximal stump; *TR*, the transplant; and *DS*, the distal stump. A section was taken through the proximal stump and the transplant for microscopic examination.

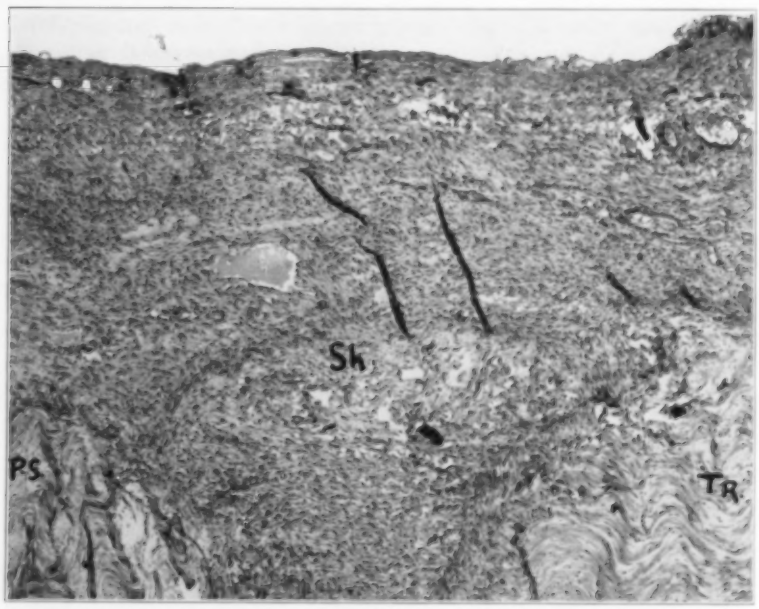


FIG. 3

Young bovine tendon preserved in homologous plasma (× 34). Section, one week after operation, shows *PS*, the proximal stump; *TR*, the transplant; and *SH*, the sheath invading the gap between the proximal stump and the transplant.

In the past, the main controversy concerning the healing of autogenous-tendon transplants has been whether the transplants have tenoblastic activity or whether they become necrotic, act as struts, and are replaced by tenoblasts from the host. Mason and Shearon reported tenoblastic proliferation in the transplant [3]. In the experiments to be reported here the process of healing associated with autogenous-tendon transplants was compared with the healing process associated with the two types of heterogenous-tendon transplants.

Tucker observed that human bone that had been preserved in plasma showed histological evidence of viability with osteogenic properties [5]. He reported that when he used human bone preserved in plasma for periods up to eleven months

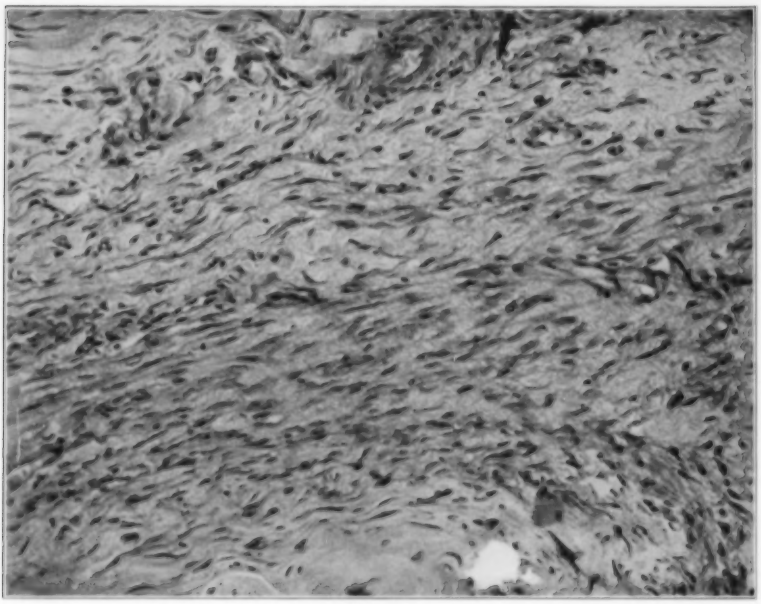


FIG. 4-A

Young bovine tendon preserved in homologous plasma (× 90). Section, one week after operation, shows that the proximal stump of the dog's tendon presented active proliferation of tenoblasts. The nuclei were larger than normal and there were many basophilic fibers.

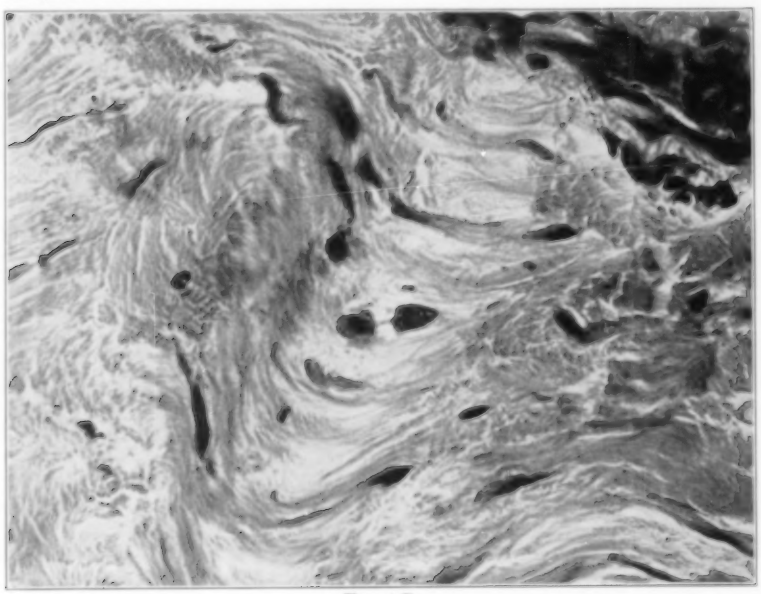


FIG. 4-B

A higher power of Fig. 4-A showed a mitotic figure in telophase in a tenoblast (× 600).

(for grafting in forty patients) there was graft union in every instance without postoperative infection.

In further experiments, Tucker reported that storage of tissue in homol-

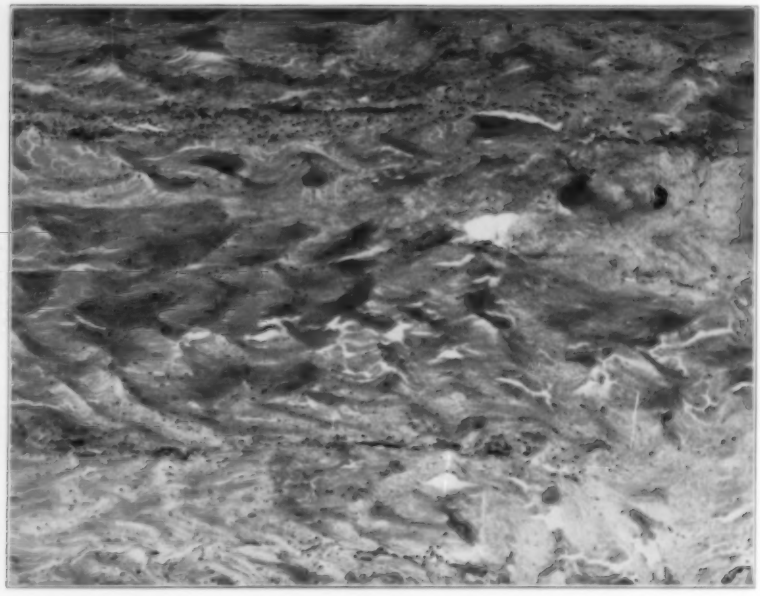


FIG. 5

Autogenous-tendon transplant (× 37). One week after operation the proximal end of the transplant showed karyorrhexis—nuclear and protoplasmic fragmentation.

ogous plasma at "a low metabolic temperature" greatly reduced its ability to induce unfavorable antibody reactions [6]. Tucker used preserved calf bone in twenty-two bone-grafting procedures performed on twenty-two human beings; there was no antigenic reaction and only one case of non-union (the latter was due to trauma) [6]. Fischer and Clayton used preserved calf bone as implants in eight human beings without resultant ill effects [2]. More recently, Clayton reported that he had used preserved calf-bone implants in sixty-one orthopaedic procedures without any infection or reaction except for one patient, an infant with a congenitally deformed tibia into which a fetal calf epiphysis was transplanted [1]. In this instance, serous fluid drained from the incision for eight weeks before spontaneous healing occurred.

PREPARATION OF THE HETEROGENOUS-TENDON TRANSPLANTS

The young bovine-tendon transplants used in Experiment 1 were obtained from the extensor carpi radialis tendons of calves, four to six weeks old, weighing 125 to 150 pounds. Government-inspected calves with negative tests for tuberculosis and brucellosis were used. Immediately after the animal was killed it was hung up by its hindfeet and bled from the jugular vein to prevent the reflux of blood into the extremities through the portal system. The tendon and other tissue to be used were removed from the calf under strict operating-room aseptic technique. All of the tendons were placed in a large container and covered with a sterile solution of 20 per cent bovine plasma in physiological saline solution. To each liter of solution were added 1,000,000 units of penicillin, 500,000 units of mycostatin, and 1.5 grams of sodium sulfathiazole. The container was sealed and stored in the refrigerator at 40 degrees Fahrenheit. After fourteen days of storage a sample of solution was cultured for the presence of both anaerobic and aerobic bacteria. If all cultures were negative, the tendons were transferred, using aseptic technique, to individual containers with fresh plasma solution. A final culture was then made of each container with thioglycollate

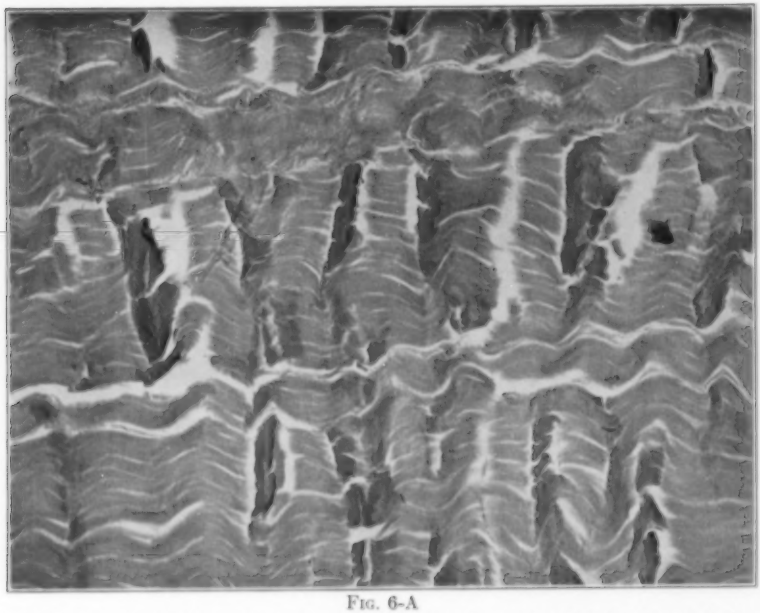


FIG. 6-A

Young bovine tendon preserved in homologous plasma (× 75). Two weeks after operation the entire transplant had undergone anuclear necrosis.

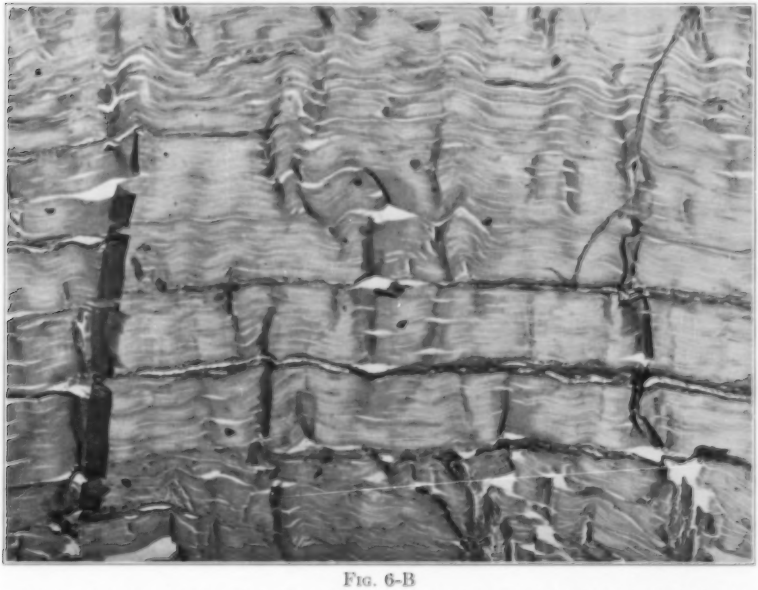


FIG. 6-B

Autogenous tendon (× 37). Two weeks after operation the entire transplant presented anuclear necrosis. The blood vessels in the septa came from the dog's stumps.

broth media. After the tendons had been kept for forty-seven days at the Medical Research Department of the Squibb Institute, they were stored at 40 degrees Fahrenheit in our laboratory for forty-five to 149 days until used. The tendon sheaths were not used in these transfers.

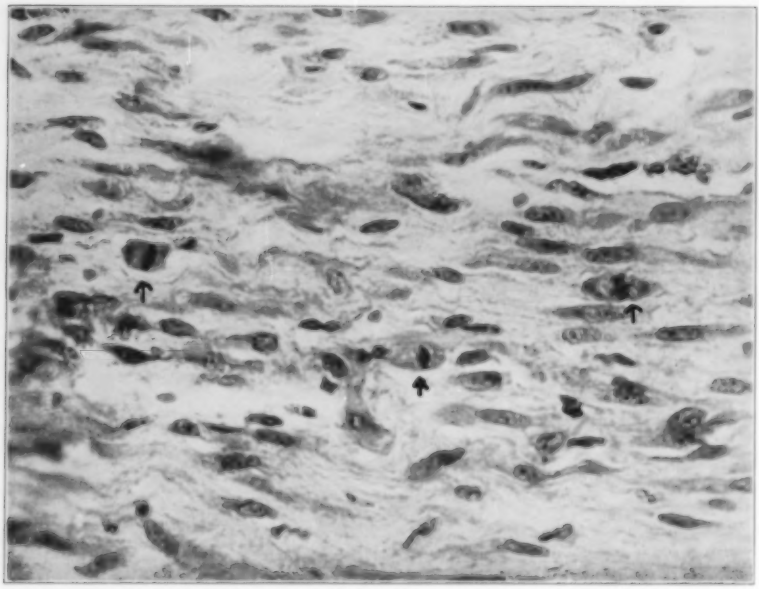


FIG. 7

Young bovine tendon preserved in homologous plasma (× 350). Two weeks after operation the proximal stump of the dog presented a very active proliferation of tenoblasts. The nuclei were larger and rounder. Arrows point to three mitotic figures in metaphase.

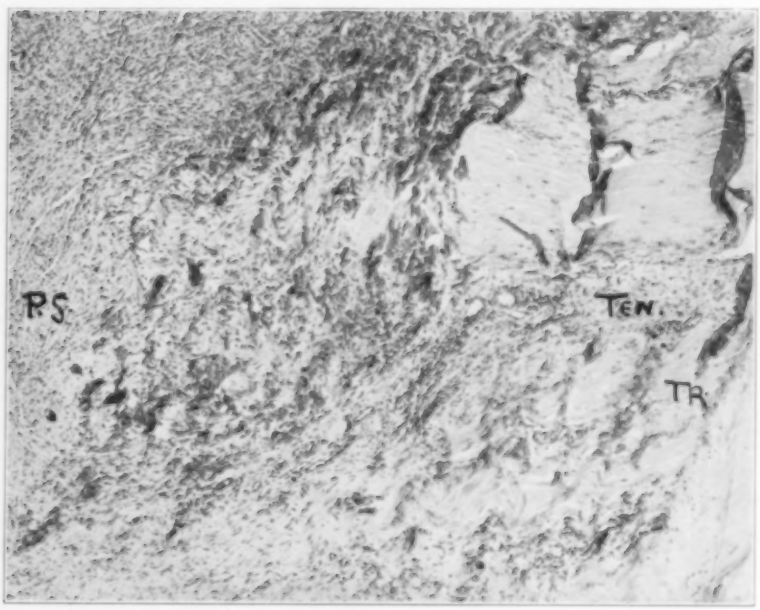


FIG. 8-A

Autogenous-tendon transplant (× 37). Two weeks after operation the tenoblasts (*TEN*) from the proximal stump (*PS*) invaded the transplant (*TR*) as finger-like projections.

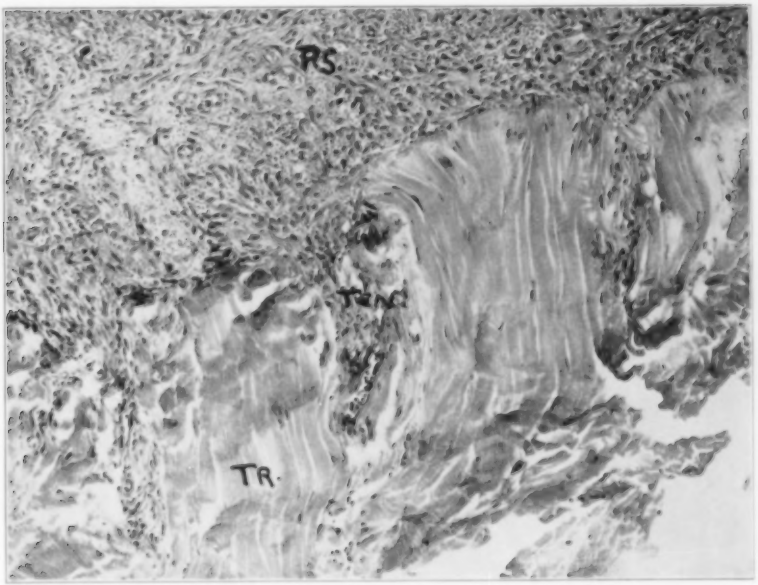


FIG. 8-B

Embryonic irradiated, frozen, bovine tendon (× 75). Two weeks after operation, the tenoblasts (*TEN*) from the proximal stump (*PS*) invaded the anuclear necrotic transplant (*TR*).

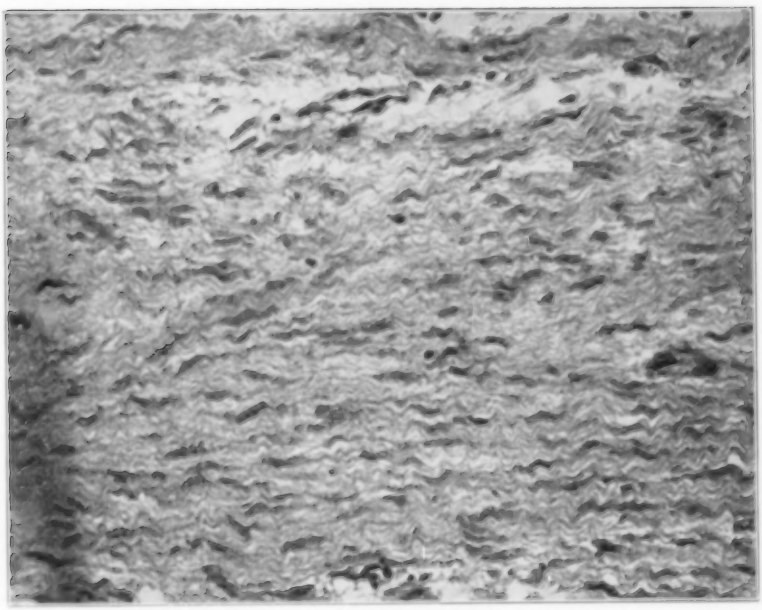


FIG. 9

Autogenous-tendon transplant (× 150). Three weeks after operation the proximal stump of the dog presented proliferating tenoblasts with wavy fibers, the first anatomical evidence of collagen.

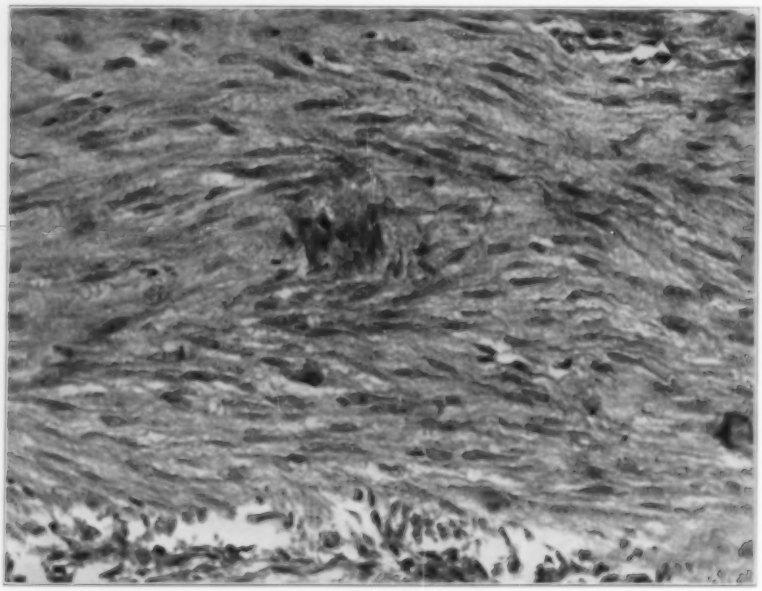


FIG. 10

Autogenous-tendon transplant (× 235). Six and one-half weeks after operation the tenoblasts that had invaded the transplant were arranged longitudinally in the line of stress.

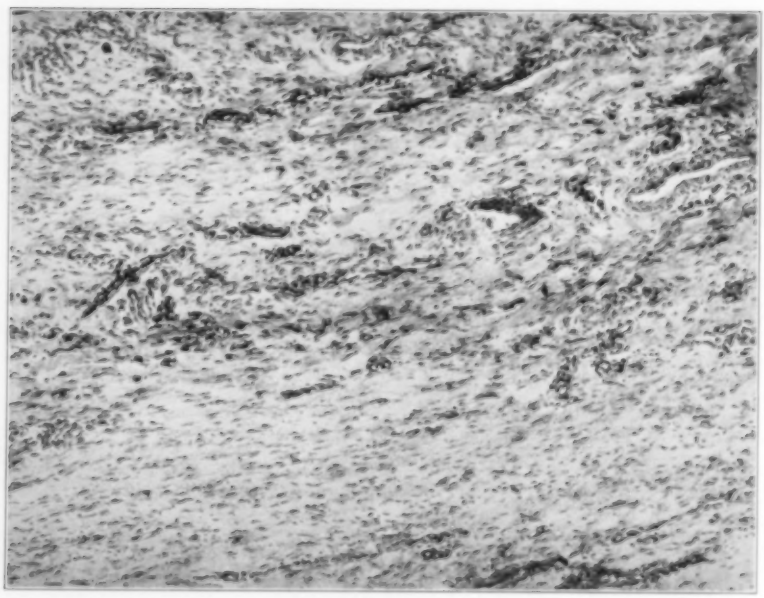


FIG. 11

Autogenous-tendon transplant (× 37). Three weeks after operation the replacement of transplant by tenoblasts from the stumps was complete.

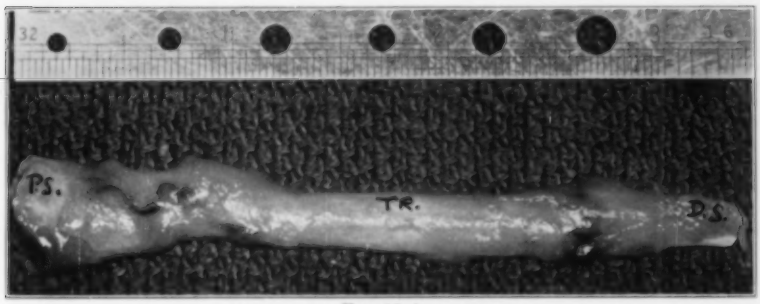


FIG. 12-A

Autogenous-tendon transfer. The gross specimen nine weeks after operation appeared viable. *PS* shows the proximal stump; *TR*, the transplant; and *DS*, the distal stump.

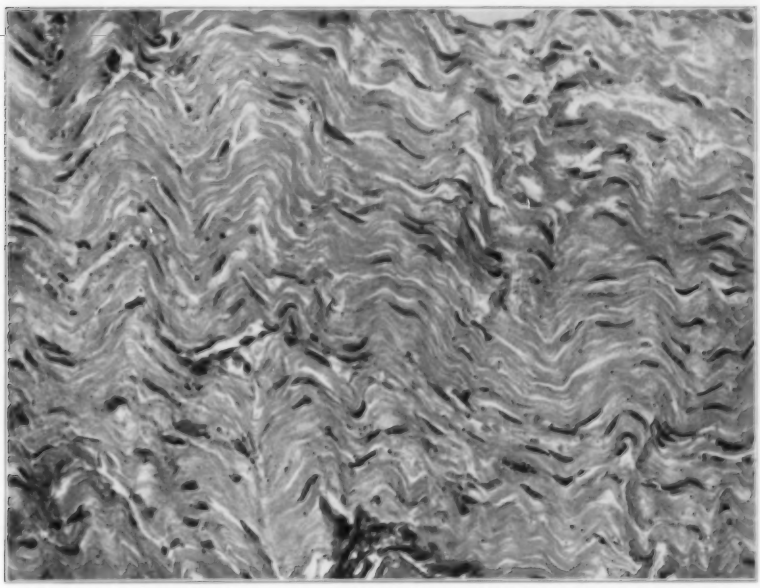


FIG. 12-B

Higher-power magnification (× 150) through center of transplant in Fig. 12-A showed complete replacement by adult tendon cells.

For Experiment 2 extensor carpi radialis tendons were obtained under sterile technique from sixth-month calf fetuses. The tendons were irradiated with 2,000,000 REP (roentgen equivalent physical units) and preserved in sterile plastic containers at —70 degrees Fahrenheit until used [4].

Thirty-three dogs were given sodium nembutal anesthesia intravenously, and a segment of the extensor carpi radialis tendon was excised and replaced by a tendon transplant. The gap thus formed was usually about one centimeter longer than the segment of the tendon excised because of retraction of the proximal stump. Most of the tendon transplants inserted were four centimeters in length, but some were as long as five centimeters. They were always sutured under tension. In some cases the transplant was slightly longer than the excised segment, but never as long as the resultant defect.

The tendons were sutured with the Bunnell silk technique using 000 silk. The sheath was not sutured in any case. After operation a plaster cast was used

FIG. 13-A

Young bovine tendon preserved in homologous plasma. Gross specimen, ten weeks after operation, appeared as one homogeneous structure. *PS* shows the proximal stump; *TR*, the transplant; and *DS*, the distal stump.

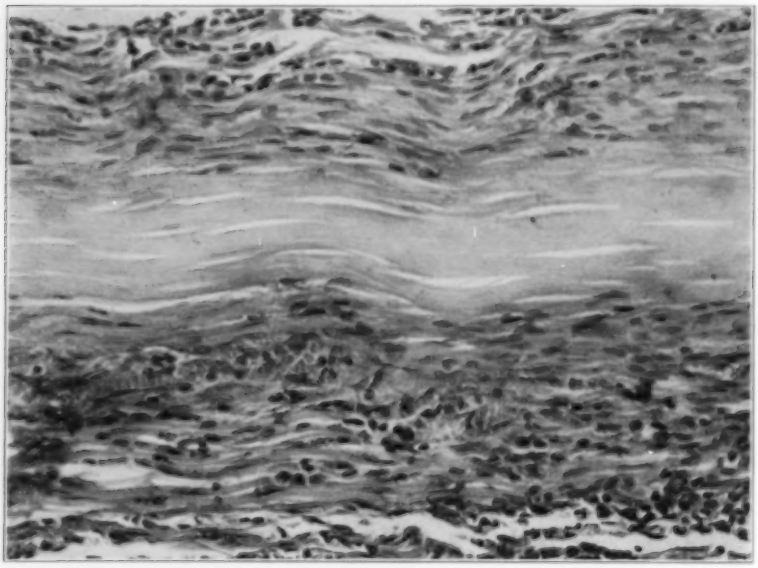


FIG. 13-B

Higher-power magnification (× 235) through the center of transplant in Fig. 13-A showed complete replacement by adult tendon cells.

for immobilization for about three weeks. In six animals, however, casts were used for less than three weeks and were removed when the animals were sacrificed. The cast maintained the humero-ulnar joint in flexion and the radiocarpal joint in extension.

In Experiment 1 twelve transplants, made by cutting six preserved bovine tendons in halves, were inserted in twelve dogs. Cultures of the bovine plasma were made at the time of transplant in each instance; all were negative. At weekly intervals from one week to 100 days after operation, the animals were sacrificed and the tendon transplant exposed. Particular note was made of the connective-tissue reaction in the tissues surrounding the graft as well as of the appearance of the tendon transplant and of the proximal and distal stumps. The tendon and attached stumps were then removed for histological examination. The tissues were fixed in 10 per cent formalin for twenty-four hours, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. For purposes of comparison microscopic sections were also prepared from the preserved bovine tendon prior to transplantation and from the dog's tendon.

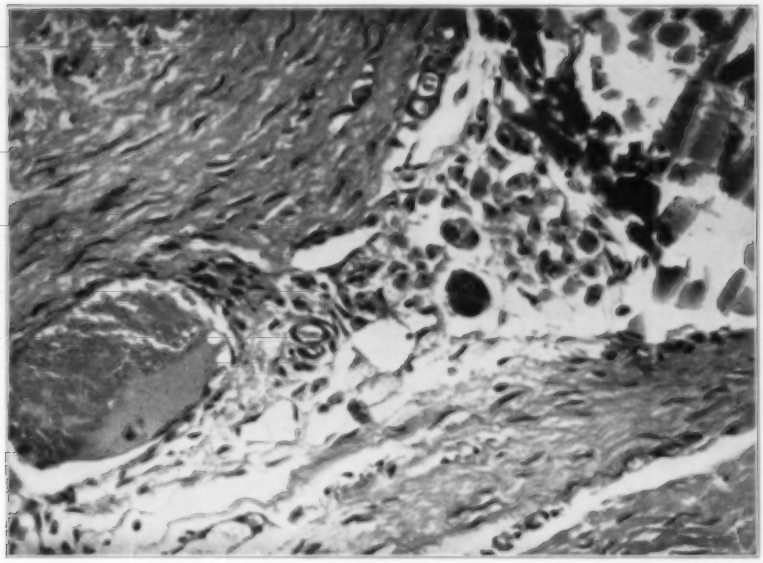


FIG. 14

Young bovine tendon preserved in homologous plasma (× 350). Seven weeks after operation the proximal stump of the dog showed a clump of plasma cells.

In Experiment 2 the same procedure was employed in nine dogs except that the transplants used were of irradiated, frozen, fetal bovine tendon.

In Experiment 3 autogenous transplants obtained from the opposite extremity at the same surgical procedure were inserted in twelve animals. The same technique as in the other two experiments was used.

RESULTS

The gross and microscopic changes observed with the three types of tendon transplant are detailed in Table I.

Some of the characteristics observed were common to all three experiments. Thus, among all the transplants examined during the first two weeks after grafting the junctions between the stumps and the transplants were united, in the main, by proliferated peritendinous tissue, and within all the stumps there was great tenoblastic activity with many mitoses and increased numbers of blood vessels. Again, all the transplants examined early showed complete ischemic necrosis, whereas all those examined late showed complete replacement of the graft by viable adult tendon.

Along with these similarities there were also differences, which were apparent in the three types of transplants. These included variations in the time required for the replacement of the transplant by tenoblasts, variations in the time of appearance of the collagen fibers and of appearance of tenoblasts oriented along the lines of stress, and variations in the time required for the formation of the structure of adult tendon. Probable antibody reactions were observed only with the heterogenous transplants that had been preserved in homologous serum.

With the autogenous and with the irradiated, frozen, heterogenous transplants wavy intercellular fibrils (the first histological evidence of collagen formation) were observed at three weeks in the tenoblasts, proliferating in the stumps and invading the transplant. On the other hand, with the heterogenous transplants preserved in homologous plasma, these fibrils did not appear until five weeks after transplantation. The tenoblasts were found to be in longitudinal

TABLE I

PROTOCOL OF EXPERIMENTS

1. Autogenous-Tendon Transplants in Dogs
2. Irradiated, Frozen, Fetal Bovine Tendon Transplants in Dogs
3. Young Bovine Tendons, Preserved in Homologous Serum, Transplanted in Dogs

Time Elapsed from Transplantation (*Weeks*)	Paratenon Proliferation	Sheath Permits Gliding	Synovial Membrane in Sheath	Proliferation of Stumps	Ischemic Necrosis of Transplant	Transplant Replacement from Stumps	Sensitivity Reaction
1	Cellular (leukocytes, lymphocytes, histiocytes, and fibroblasts) and vascular in gaps, around stumps, and transplants in 1, 2, and 3	No in 1, 2, and 3	None in 1, 2, and 3	Tenoblastic and vascular in 1, 2, and 3	Karyorrhexis and anuclear necrosis	None in 1, 2, and 3	None in 1, 2, and 3
2	Fibroblastic and vascular, twice as great; other cellular reaction half as great, as at 1 week around stumps and transplants in 1, 2, and 3	No in 1, 2, and 3	None in 1, 2, and 3	Tenoblastic and vascular, about twice as great as at 1 week in 1, 2, and 3	Ischemic necrosis complete in 1, 2, and 3	Tenoblasts and blood vessels from stumps invade ends of transplants in 1, 2, and 3	None in 1, 2, and 3
3	Fibroblastic and vascular, greater than at 2 weeks, about stumps and transplants in 1, 2, and 3; other cellular reaction subsided	Yes in 1, 2, and 3	None in 1, 2, and 3	Stumps completely replaced by tenoblasts and blood vessels in 1, 2, and 3; wavy collagen fibers in 1 and 2 only		Tenoblasts from stumps completely replace transplant in 1; replace half of transplant in 2; replace one-third of transplant in 3; wavy fibers in 1 and 2 only	None in 1, 2, and 3

4	Fibroblastic and adult fibrous tissue about equal, over stumps and transplants in 1, 2, and 3, and greater than at 3 weeks	None in 1, 2, and 3	Same as at 3 weeks with wavy collagen fibers in 1 and 2	Tenoblasts from stumps completely replace transplant in 1; replace two-thirds of transplant in 2; replace one-half of transplant in 3; wavy fibers in 1 and 2 only	None in 1, 2, and 3
5	Mature fibrous sheath formed around stumps and transplants	Over proximal stump in 3 only*	Increasing number of wavy fibers in 1 and 2; wavy fibers first seen in 3	Tenoblasts with wavy fibers from stumps completely replace transplant in 1; replace three-quarters of transplant in 2; replace two-thirds of transplant in 3	None in 1, 2, and 3
6		None in 1, 2, and 3	Collagen fibers oriented along stress lines in 1 only	Tenoblasts with wavy fibers from stumps completely replace transplants in 1 and 2; replace two-thirds of transplant in 3	None in 1, 2, and 3
7		None in 1, 2, and 3	Collagen fibers oriented along stress lines in 1 and 2	Tenoblasts with wavy fibers from stumps completely replace transplants in 1 and 2; replace two-thirds of transplant in 3	Plasma cells in clumps in 3 only
8		None in 1, 2, and 3	Collagen fibers oriented along stress lines in 1, 2, and 3	Tenoblasts with wavy fibers and collagen fibers oriented along stress lines from stumps completely replace transplants in 1 and 2; replace two-thirds of transplant in 3	Rheumatoid nodules in proximal stump and transplant in 3 only

TABLE I *(Continued)*

PROTOCOL OF EXPERIMENTS

1. Autogenous-Tendon Transplants in Dogs
2. Irradiated, Frozen, Fetal Bovine Tendon Transplants in Dogs
3. Young Bovine Tendons, Preserved in Homologous Serum, Transplanted in Dogs

Time Elapsed from Transplantation (*Weeks*)	Paratenon Proliferation	Sheath Permits Gliding	Synovial Membrane in Sheath	Proliferation of Stumps	Ischemic Necrosis of Transplant	Transplant Replacement from Stumps	Sensitivity Reaction
9			Over distal stumps in 3 only*	Stumps completely replaced in 1 by adult tendon; in 2 and 3, one-half by adult tendon and one-half by tenoblasts with wavy fibers		Complete replacement of 1 by adult tendon; complete replacement of 2 and 3, one-half by adult tendon and one-half by tenoblasts with wavy fibers	None in 1, 2, and 3
10			Over both stumps in 3 only*	Replacement by adult tendon in 1, 2, and 3		Complete replacement of transplants by adult tendon in 1, 2, and 3	None in 1, 2, and 3
11			Over both stumps in 3 only*				None in 1, 2, and 3
14¼			Over both stumps with ganglion over distal stump in 3 only*				None in 1, 2, and 3

*Synovial membrane was seen only in Experiment 3. The reason why synovial membrane was not seen in Experiments 1 and 2 is unknown.

TABLE II

Experiment	Time of Appearance of Collagen in Stumps and Transplants* (*Weeks*)	Time of Appearance of Stress Lines in Stumps and Transplants** (*Weeks*)
1. Autogenous transplant	3	6½
2. Heterogenous, irradiated, frozen, transplants	3	7½
3. Heterogenous and homologous transplants	5	8

*Wavy fibers in tenoblasts.
**Tenoblasts appear longitudinally.

TABLE III

REACTIONS IN THE THREE EXPERIMENTS

Similar Reactions	Different Reactions
Peritendinous proliferation	Time of appearance of collagen in tenoblasts
Proliferation of stumps; mitoses	
Ischemic necrosis of transplants	Time of appearance of stress lines with tenoblasts
Tenoblastic invasion of transplants from stumps	Time of replacement of transplants by tenoblasts and adult tendon cells
Replacement of transplants by tenoblasts and adult tendon cells	Probable antibody reactions
	Synovial membrane in sheaths

arrangement, with their long axes oriented along lines of stress in the stumps and transplants at six and one-half, seven and one-half, and eight weeks, respectively, in the autogenous transplants (Fig. 10), in the heterogenous, irradiated, frozen transplants, and in the heterogenous transplants preserved in homologous serum.

Complete replacement of the transplants by tenoblasts growing in from the two stumps occurred in three weeks in the autogenous transplants (Fig. 11), in six weeks in the heterogenous irradiated transplants, and in nine weeks in the heterogenous transplants preserved in homologous serum. Grossly, all the stumps and transplants (except for the sutures), regardless of type, appeared as homogeneous structures at about ten weeks. The tenoblasts were replaced by adult tendon cells by nine weeks in the autogenous grafts, and by ten weeks in each of the heterogenous implants.

The only histological evidence to suggest an antibody reaction was observed

FIG. 15-A

Young bovine tendon preserved in homologous plasma. Eight weeks after operation the gross specimen presented some liquefaction, but the culture produced no growth.

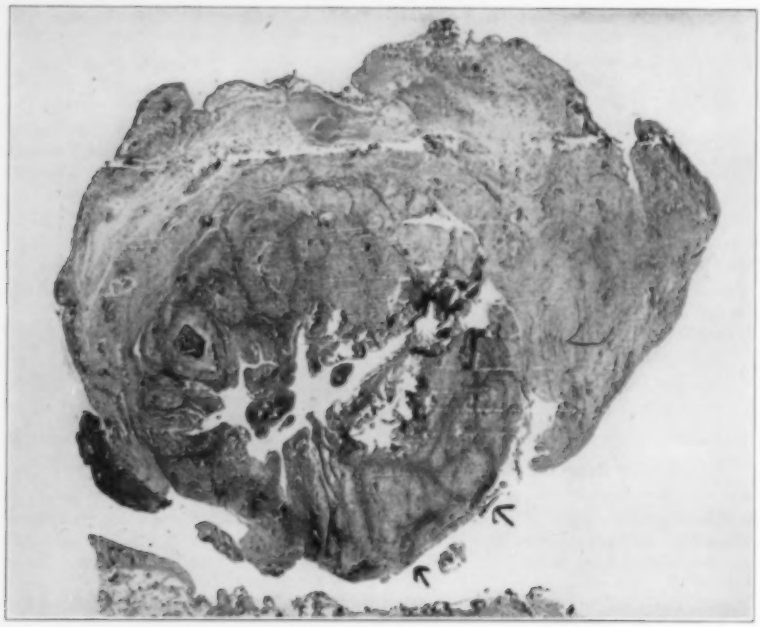


FIG. 15-B

Section (× 39) taken through the center of the transplant in Fig. 15-A. The arrows point to rheumatoid-like nodules.

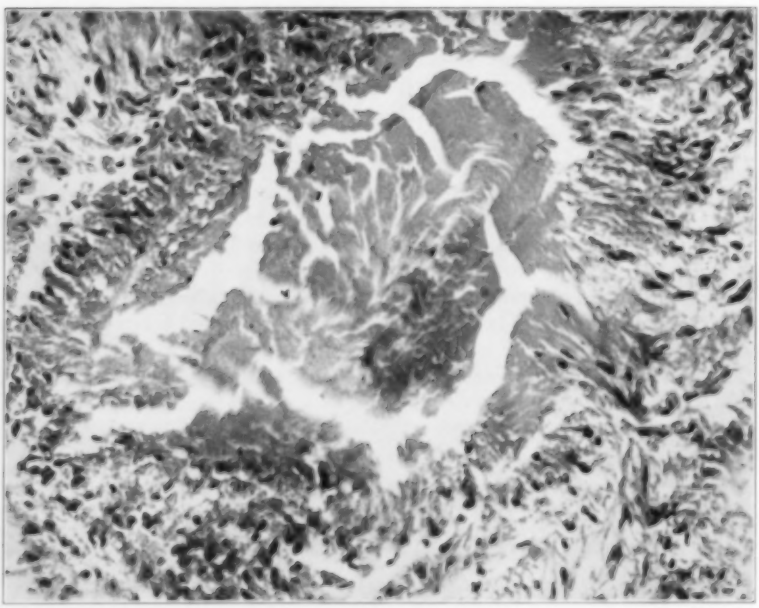


FIG. 15-C

Rheumatoid-like nodules magnified (× 350) showed collagen necrosis in the center and plump fibroblasts in the periphery.

with the transplants of young bovine tendons that had been preserved in homologous serum. At seven weeks the proximal stump showed some clumps of

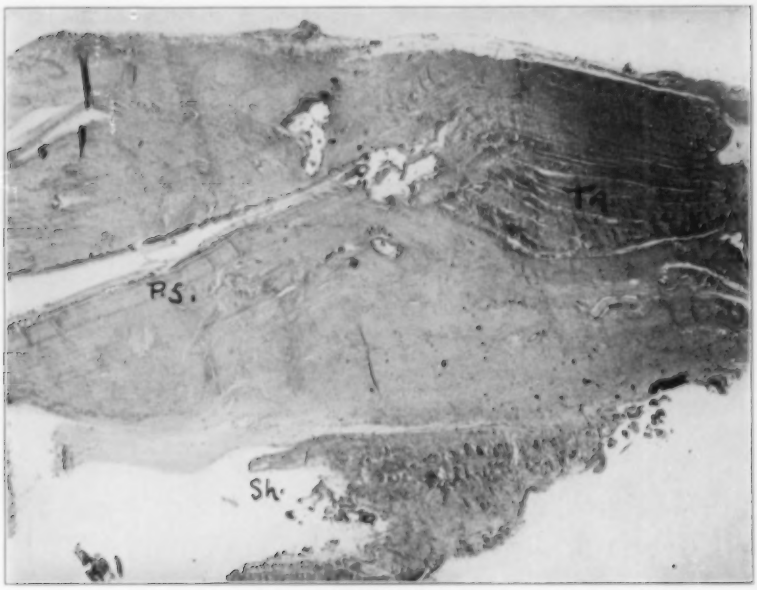


FIG. 16-A

Young bovine tendon preserved in homologous plasma (× 37). Five weeks after operation the sheath (*SH*) over the proximal stump (*PS*) had a synovial lining. *TR* is the transplant.

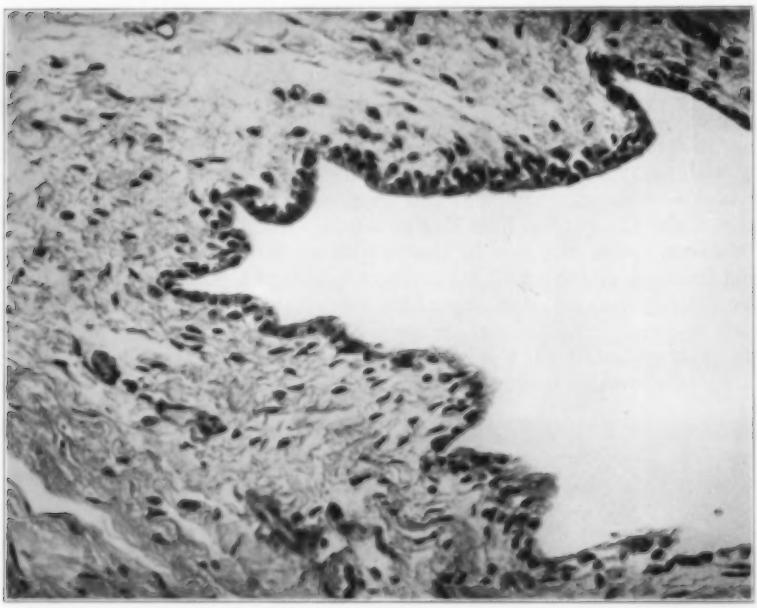


FIG. 16-B

Higher power (× 350) of sheath in Fig. 16-A showed endothelial lining of the synovial membrane.

plasma cells (Fig. 14). At eight weeks, the proximal stump and transplant showed areas with rheumatoid-like nodules (Figs. 15-A, 15-B, and 15-C). However, in both of these animals active, progressive tenoblastic proliferation was noted.

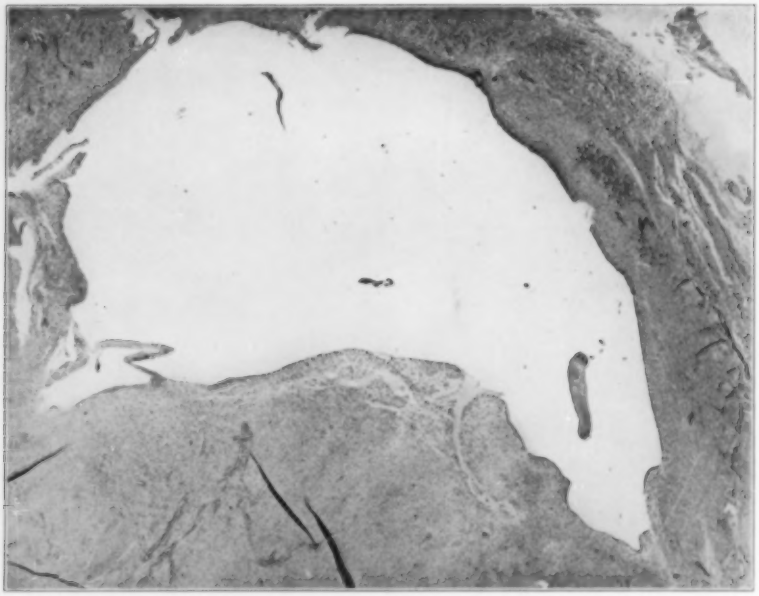


FIG. 17

Young bovine tendon preserved in homologous plasma (× 37). One hundred days after operation the sheath over the proximal stump formed a bursa.

DISCUSSION

Our findings with autogenous-tendon transplants in dogs were comparable to the findings of Mason and Shearon in the following respects. During the first two weeks the union between stumps and transplants was effected mainly by proliferation of the sheath tissues. The stumps presented great tenoblastic activity with many mitoses and an increase in blood vessels. The time of appearance of the collagen fibers and the time required for the orientation of the collagen fibers along stress lines was about the same in both experiments. Mason and Shearon found evidence in their experiments of tenoblastic activity originating in some of the small tendon transplants. In our experiments no tenoblastic activity was found in any of the transplants. Instead, complete ischemic necrosis occurred in all of our transplants followed by invasion of the transplants by tenoblasts from the host's tendons. Eventually, complete replacement of transplants by adult tendon occurred.

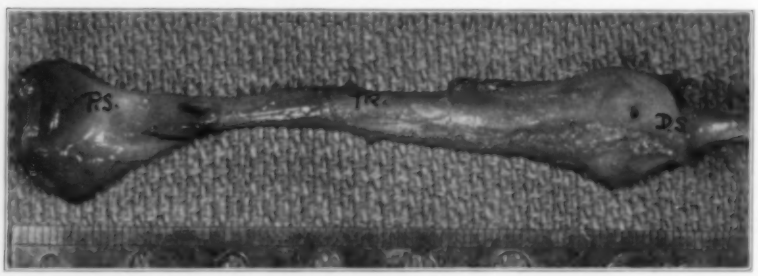


FIG. 18-A

Young bovine tendon preserved in homologous plasma. The gross specimen one hundred days after operation appeared viable and homogeneous. *PS* shows the proximal stump; *TR*, the transplant; and *DS*, the distal stump.

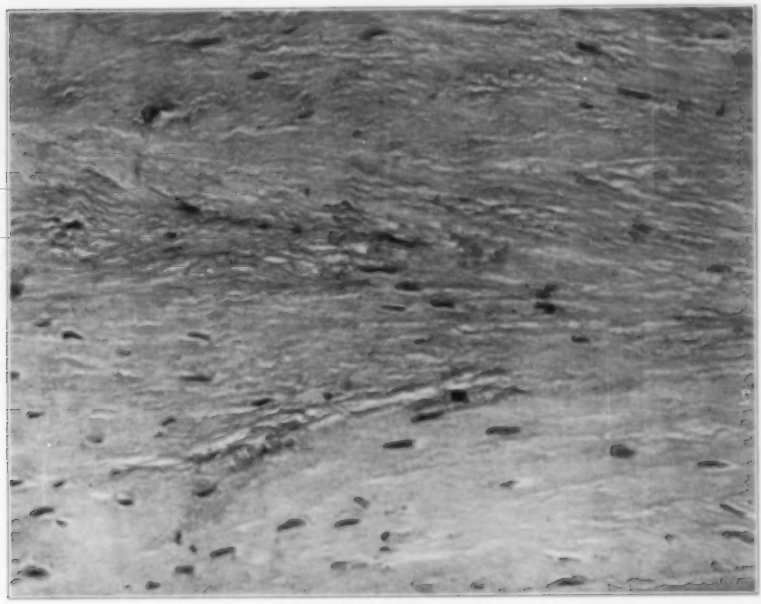


FIG. 18-B

Higher power (× 115) through center of the transplant in Fig. 18-A revealed normal adult tendon similar in structure to young bovine tendon and to dog tendon.

Our findings with young heterogenous bovine tendons, preserved in homologous plasma and transplanted in dogs, differed from those of Tucker [6], who used heterogenous bovine bone preserved in homologous plasma. He found that the bone preserved in homologous plasma behaved as a living tissue and proliferated when transplanted into another species, whereas in our experiments with young bovine tendon similarly preserved, ischemic necrosis occurred in all cases. In our experiments, the fact that a probable antibody reaction was found only with tendons that had been preserved in homologous plasma suggests the possibility that the antigen was a protein from the homologous plasma.

CONCLUSION

Young heterogenous bovine tendons preserved in homologous plasma and irradiated, frozen, fetal bovine tendons transplanted in dogs cause reactions which differ only slightly from those associated with autogenous-tendon transplants in dogs. All transplants underwent necrosis but acted as struts, which were replaced by tenoblasts from the stumps of the host.

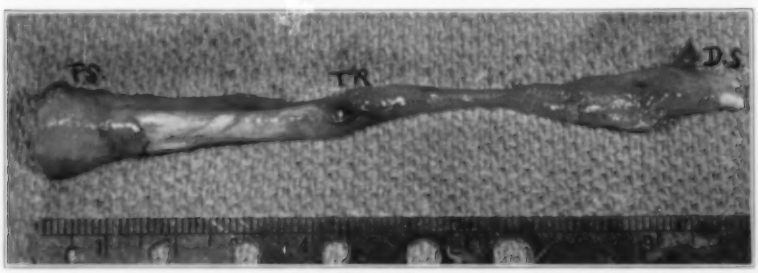


FIG. 19-A

Autogenous-tendon transplant. The gross specimen one hundred days after operation appeared homogeneous. *PS* shows the proximal stump; *TR*, the transplant; and *DS* the distal stump.

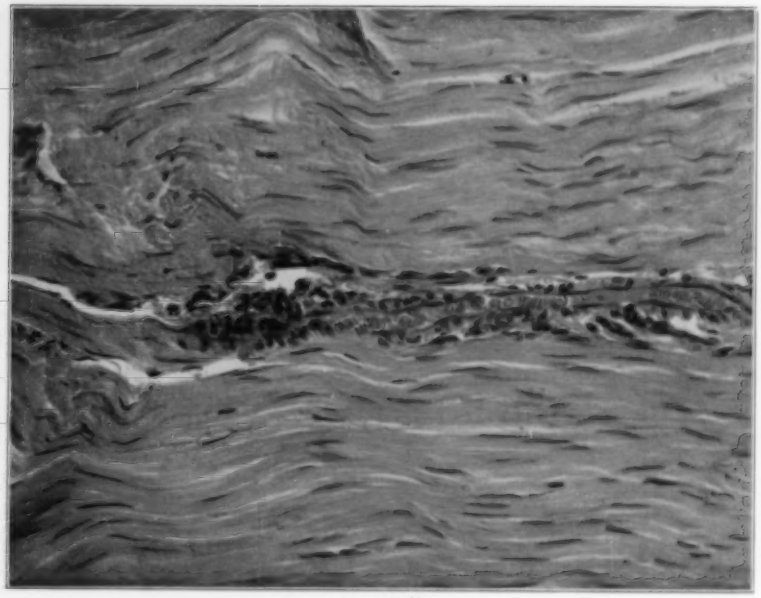


FIG. 19-B

Higher power (× 150) through the center of the transplant in Fig. 19-A revealed adult tendon similar to structure in Fig. 1 except for the blood vessel in the center.

REFERENCES

1. CLAYTON, IRVIN: Cultured Calf Bone; a New Bone Grafting Material. Illinois Med. J., **110**: 49-52, 1956.
2. FISCHER, W. B., and CLAYTON, IRVIN: Surgical Bone Grafting with Cultured Calf Bone. Quart. Bull. Northwestern Univ. Med. School, **29**: 346-349, 1955.
3. MASON, M. L., and SHEARON, C. G.: The Process of Tendon Repair. An Experimental Study of Tendon Suture and Tendon Graft. Arch. Surg., **25**: 615-692, 1932.
4. MEEKER, I. A., JR., and GROSS, R. E.: Low-Temperature Sterilization of Organic Tissue by High-Voltage Cathode-Ray Irradiation. Science, **114**: 283-285, 1951.
5. TUCKER, E. P.: The Preservation of Living Bone in Plasma. Surg., Gynec., and Obstet., **96**: 739-749, 1953.
6. TUCKER, E. J.: Studies of the Use of Cultured Calf Bone in Human Bone Grafts. Clin. Orthop., **7**: 171-188, 1956.

DISCUSSION

DR. MICHAEL MASON, CHICAGO, ILLINOIS: The work that Dr. Flynn and his associates have done corroborates, to a large extent, the experiments which Dr. Shearon and I carried out some years ago, and the experimental work which Dr. Harvey Allen and I carried out.

I want to take issue with one point—whether these grafts are viable. It is extremely difficult in studying these microscopic sections to determine the exact source of the proliferating cells. We felt that in many instances there was evidence of proliferation in the ends of the grafts and that many of these were bundles of tendon cells. It is a question, of course, whether these are specific cells. I am glad that Dr. Flynn and his associates feel, as we did, that the tendon actually takes part.

The other question is the vitality of the grafts. We felt, in going over many sections of the tendons, that the graft may not remain entirely viable but that there were sections that did remain viable, and that smaller grafts of lesser diameter remained largely viable and carried over to the end as viable tissue.

DR. E. E. PEACOCK, JR., CHAPEL HILL, NORTH CAROLINA: In addition to certain clinical applications, the work done by Dr. Flynn and his associates represents a contribution to our basic knowledge in the general field of tissue transplantation. Tendons are relatively simple structures; they contain only three elements—collagen, ground substance, and fibrocytes. Thus, they offer an unusual opportunity to study relatively simple preparations of these three substances after heterologous and homologous transplantations. Dr. Flynn has shown us the

(Continued on page 137)

Adductor Myotomy and Obturator Neurectomy for the Correction of Adduction Contracture of the Hip in Cerebral Palsy*†

BY HENRY H. BANKS, M.D., AND WILLIAM T. GREEN, M.D., BOSTON, MASSACHUSETTS

From the Department of Orthopaedic Surgery, Harvard Medical School, and the Children's Medical Center, Boston

Spasticity of the adductor muscles of the hip is a common disability in cerebral palsy. Adduction of the thighs and scissoring interfere with balance, gait, and even nursing care. Coxa valga and dislocation of the hip are not infrequently observed. Yet, the value of surgical procedures in the treatment of this disability has been the subject of considerable discussion.

During the past year we have continued the evaluation of all patients with cerebral palsy treated by orthopaedic surgical procedures at the Children's Hospital, Boston, Massachusetts, in the period from 1940 to 1956. Adduction contracture was found to be the most common hip deformity. Among all the deformities treated it was second only to equinus deformity of the foot.

CLINICAL DATA

During the period 1940 to 1956 eighty-nine patients were operated on to correct adduction contracture of the hip. Of these patients, sixty-five were recently seen and evaluated in a follow-up clinic. Sufficient data are available in the records of nine additional patients to adequately determine the end result of their surgical procedures, although they were not seen during the recent evaluation. There are no data available concerning the end result in fifteen patients. All the patients analyzed in this study were seen more than one year (an average of six years) after their operations. Forty-two were seen more than five years after surgery, and twenty-two of these were evaluated ten to seventeen years after operation.

All eighty-nine patients in this series had the spastic type of cerebral palsy except for three who had athetosis as well. All were under eighteen years of age at the time of surgery, and forty-six were five years old or less. The youngest patient was one year and four months of age. There were fifty male and thirty-nine female patients. Twenty-one of the patients were considered to be of normal mentality; sixty-four were definitely retarded, including twenty-eight patients who were classified as severely retarded or feeble-minded. The latter patients had an intelligence quotient of 50 or less. No data were available regarding the mentality of four patients.

There were two instances of hemiplegia, eight of paraplegia, and seventy-nine of quadriplegia. A large number of the quadriplegic patients had very severe involvement. Many had difficulty with balance, frequently because of severe scissoring. Forty-eight patients, twenty-nine of whom were more than five years of age, were not walking before surgery.

All but two patients presented very severe adduction contractures of the hips. Before operation, in seventy of the eighty-nine patients a mild-to-severe degree of flexion deformity of the hip was recorded in association with adduction contracture. All individuals with severe adduction deformities with scissoring

* Read in part at the Annual Meeting of the American Academy for Cerebral Palsy, Chicago, Illinois, November 19, 1956.

† This study was aided by a grant from the United Cerebral Palsy Research and Education Foundation.

presented some tendency, usually mild, toward an internal-rotation deformity, especially when standing. In only fifteen patients did there appear to be a marked internal-rotation contracture.

Although the adductor muscles were uniformly graded as good to normal in strength, in most instances it was very difficult to assess accurately the strength of the abductor muscles of the hips before operation. Inasmuch as a fair abductor muscle is defined as one which can carry the hip through a full range of abduction against gravity, the abductor muscles in these patients were usually graded as poor or less in strength since the adduction contracture prevented these muscles from moving the hip through a range of abduction.

The incidence of abnormalities of the hip joints in these patients was high. Severe valgus deformity of the femoral neck was very common. Among the 175 hips which came to surgery, eight showed frank dislocation, and thirty-four showed some degree of subluxation. Preoperative roentgenograms were available on only ninety-six of these hips, but it is likely that frank dislocation or severe subluxation would have been recognized clinically prior to surgery in those who did not have roentgenograms. It is possible, of course, that lesser degrees of subluxation were not recognized.

Hips were classified as dislocated when, on the roentgenograms, the head was not in contact with the acetabulum and was obviously above the level of the acetabulum. The classification of subluxation, for the purposes of this study, was used for hips in which the head was in contact with the acetabulum but to a lesser degree than normal.

In several patients in whom subluxation or dislocation of the hip later developed, roentgenograms that had been made in early infancy were available which showed normal-appearing hips which were well seated. In many of the dislocated hips of older children the acetabulum was shallow; this was interpreted as due to underdevelopment secondary to the long-standing dislocation.

TREATMENT

The majority of the patients reported in this series received a variable but usually extended period of conservative treatment prior to surgery. This included active and passive exercises which, in general, were directed toward correcting the adduction, flexion, and internal rotation of the hip. Emphasis was placed on development of active control of the hip and, in particular, on controlled abduction in gait. A certain number of patients had worn night casts to prevent contractures. These conservative measures were successful in the treatment of adduction contractures of the hip of mild degree.

If, after conservative treatment, muscle imbalance and contractures persisted, surgery was advised to correct both fixed and hypertonic deformities, provided that their release would improve function. The demonstration of some evidence of satisfactory balance was usually a prerequisite for surgical treatment. However, in certain instances, such as the presence of an adduction contracture associated with a subluxated or dislocated hip, surgery was performed prior to any demonstrable evidence of satisfactory sitting, standing, or walking balance.

Since many of the patients had severe involvement and, as might be expected, had multiple deformities, other procedures directed toward the correction of these deformities were necessary in this group. In all, 196 other procedures were performed on these eighty-nine patients.

One hundred and seventy-five procedures were performed for adduction contracture of the hip on these eighty-nine patients. Of these procedures, 171 were adductor myotomies, all but fourteen of which included neurectomy of the anterior branch of the obturator nerve. In four procedures obturator neurectomy dividing the anterior branch was performed without myotomy. In all but five of these patients the procedures were bilateral.

Adductor myotomy was performed as an open procedure in which the adductor longus, the adductor brevis, the gracilis, and, frequently, the anterior portion of the adductor magnus were divided. The extent of the division depended on how much contracture was present. Occasionally, only the adductor longus, or the adductor longus and brevis, were cut.

OPERATIVE PROCEDURE

The technique of open adductor myotomy has been used for many years at the Children's Hospital. The advantage of the open procedure as contrasted with subcutaneous tenotomy was discussed by Bradford in 1890 on the basis of his experience with this procedure at this hospital [2]. Generally, a particular technique of operation was used in this series of cases, except for some variation in detail.

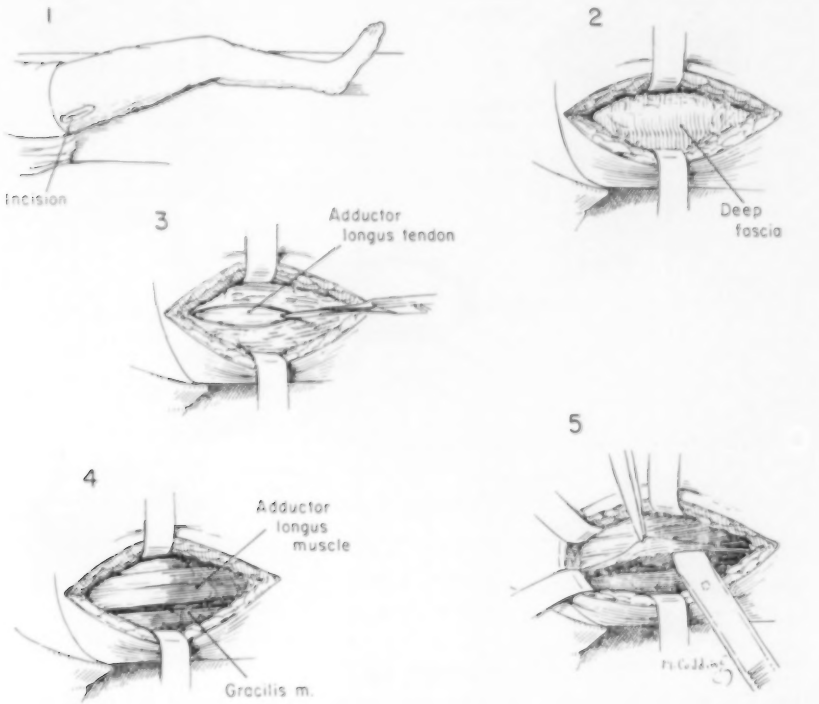


FIG. 1-A

A longitudinal incision is made over the course of the adductor longus, starting one finger-breadth distal to the pubis. The adductor longus is defined, and the interval between it and the adductor brevis is developed.

With the patient in the supine position and with both legs adequately draped free to allow manipulation, a longitudinal incision is made over the course of the adductor longus, beginning about one-half inch below the pubis and extending downward for three to four inches (Fig. 1-A). The incision is developed by sharp dissection down to the fascial covering over the adductor longus, which is then incised and cleared from this muscle. Before the adductor longus is sectioned, the interval between the adductor longus and brevis is defined medially so that the branches of the anterior portion of the obturator nerve may be visualized. The branches to the adductor longus, brevis, and gracilis are usually located and positively identified by gentle pinching with a forceps so as to stimulate contraction of the muscles (Fig. 1-B). These branches

are sectioned and dissected proximally to permit the removal of approximately an inch of the nerve. When this is accomplished, the adductor longus is cut in its tendinous portion close to its origin from the pubis. The adductor brevis is then sectioned in an oblique fashion to minimize the extent of the dead space, which is much larger if the muscle is divided transversely at the same level as the longus. If the gracilis is contracted, as may be demonstrated at this point in the procedure by abduction of the hip, this muscle is sectioned by a long oblique cut extending distally from the pubis (Fig. 1-C).

The tightness of the persisting muscles may be checked at this time by abduction of the hip. It is common practice when tightness persists to divide the

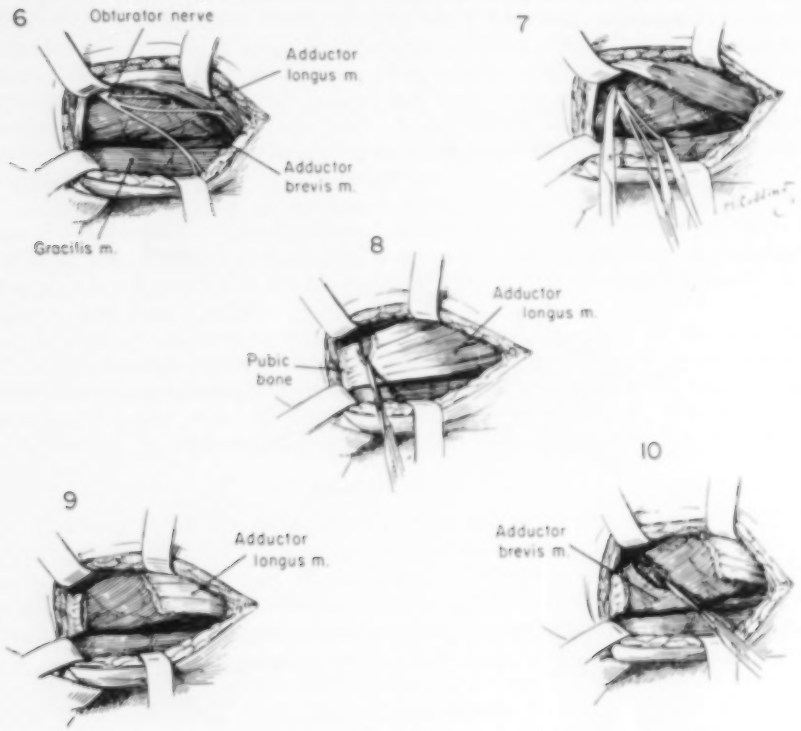


FIG. 1-B

The branches of the anterior obturator nerve to the adductor longus and brevis and the gracilis are defined and sectioned. The gracilis is sectioned, and the adductor longus and brevis are then sectioned.

most anterior fibers of the adductor magnus (Fig. 1-C). The division of the adductors is ordinarily surprisingly bloodless, provided that the vessels running in relation with the intermuscular septa are controlled before the muscles are divided. If the muscles have been divided obliquely and at different levels, very little dead space remains. The wounds are closed by approximating the subcutaneous tissues and skin only.

In patients with involvement of both lower extremities, the procedure on the two sides is performed simultaneously. The posterior portion of the obturator nerve is never cut. In no instance is the obturator nerve divided by the intra-abdominal approach. In twenty-nine instances, other procedures were performed at the same time as the operation for adduction contracture of the hip. These included twenty-seven heel-cord lengthenings and two hamstring lengthenings.

POSTOPERATIVE CARE

The postoperative management varied more than the operative technique in this series with respect to the duration of immobilization, the type and timing of exercises, the time when the patient was allowed to walk, and the type of instruction in gait. In many patients the details of the postoperative management were not well recorded. Postoperative care was necessarily affected by the motor status of the individual, and in certain instances it was modified by other operative procedures. The most common and also the recommended plan of postoperative care was as follows:

The patient was always immobilized after operation with the legs widely abducted, usually in an adequately padded, bilateral, long, hip spica (Fig. 1-C). In many instances, however, the immobilization was accomplished by bilateral, bivalved, toe-to-groin casts joined by an abduction bar. Where subluxation or

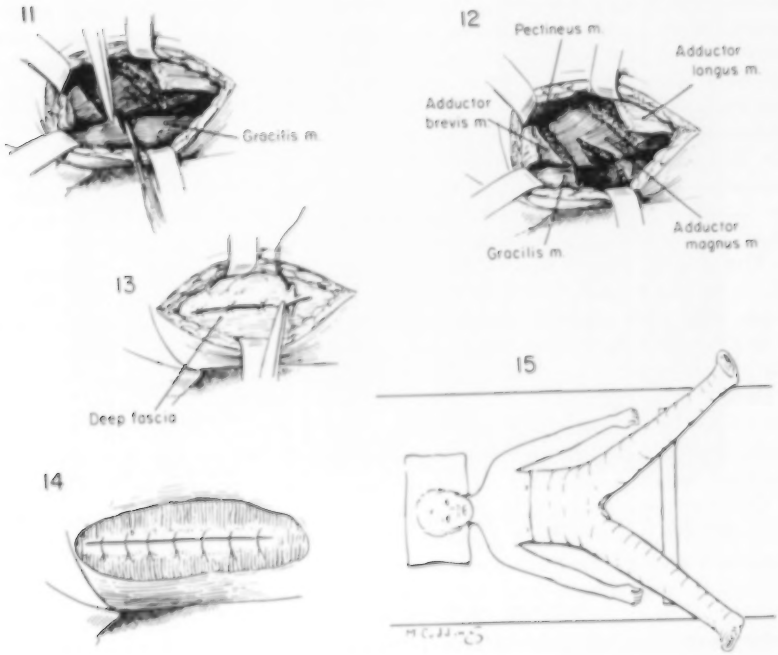


FIG. 1-C

Where tightness persists, the adductor magnus is partially but never completely divided. The skin and subcutaneous tissues are closed. The patient is immobilized in a plaster hip spica, with the legs widely abducted.

dislocation of the hip existed before operation, the spica was always used and, in fact, it was generally preferred. Where considerable hip flexion contracture was present, however, bivalved toe-to-groin casts joined by an abduction bar were helpful in that they permitted gradual stretching of this deformity after operation.

The casts were usually bivalved in the operating room. Seven to ten days after operation they were relined and fitted with straps to be used as bivalved removable splints. On the average, within one to two weeks after operation, active abduction and adduction exercises were begun. In the presence of subluxation or dislocation of the hip, exercises were postponed for as long as three months, and then all exercises for a considerable period were performed in

abduction. The time when exercises were instituted and the cast was lined varied with the motor status and the cooperation of the patient, but in any event the patient was kept in the bivalved cast at all times except during his exercise periods. At first, active assisted motions of the hip over a small range were performed with the patient in one-half of the cast. The exercises were usually performed in two or three sessions a day, with increasing ranges and activity as competence developed. Early, guided active exercises were given to teach the patient to localize muscular action; particular emphasis was placed on the function of the abductors, and the other actions were gradually added. The exercises for the abductors were at first performed in the supine position. As these muscles became more effective, they were made to work first against gravity, then against resistance, and, finally, in stance. Gentle stretching to maintain the various ranges of motion was added as indicated. Attention was directed not only toward developing and maintaining the range of abduction and the function of the abductor and adductor muscles, but also toward stretching whatever hip flexion, hamstring, heel-cord, or other contractures were present. Approximately six to eight weeks after operation, standing was begun under supervision, at first with stable support and then with crutches. Parallel bars were used when indicated. After good balance was established, which sometimes required another few weeks, gait-training with crutches was begun. The crutches were used to enable the patient to walk with as normal a gait as possible. If balance was good, crutches were used until a good gait pattern was established, which often required six months or longer. Quadriplegic patients have to use crutches much longer in their gait-training program. For patients with severe involvement, crutches may be needed indefinitely.

The length of time that casts were worn after operation was variable. At first, a bivalved abduction spica was worn continuously except for exercise periods, but, after a month, increasing periods of freedom from the cast during the day were recommended. At about six weeks after operation, the cast was worn just for rest periods and at night and then, later, only at night. Very often the abduction spica was used at first and then replaced by bivalved toe-to-groin casts joined by an abduction bar. Casts were always used at night for many months and even for several years, particularly in the young. The time when casts were removed depended on the tendency toward recurrence of contracture and the effectiveness of active abduction, both in recumbency and stance. Even after casts were removed the patients were checked regularly for recurrence of contracture and splinted again, if necessary.

METHOD OF END-RESULT EVALUATION

In cerebral palsy it is difficult to evaluate the result of treatment for a variety of reasons which have already been described by us [1]. There are many variables in each patient, some of which are not easy to classify, including the degree and type of motor involvement, the extent of deformity, and the mental status. In evaluation of surgical results consideration must be given to the natural history of the disease and any improvement that might occur without surgery. Even under the best of circumstances, the results obtained represent relative improvement rather than restoration of normal status.

In order to express the results more effectively, two classifications of the result were employed. One was a record of improvement, which compared the final result with the preoperative status of the patient; the other was a grading of the functional performance of the hip at the time of the last examination as related to the normal.

In the grading of improvement there were five classifications: namely, no improvement, 1+, 2+, 3+, and 4+ improvement. The greatest improvement recorded was that of 4+.

In the grading of the functional performance the classification depended

FIG. 2-A

Before operation. Spastic paralysis in C.S., at the age of five years and eight months in March 1956.

upon the function of the hip at the time of the final examination and did not consider the status before operation. Factors in this grading were the range of abduction, the adductor-abductor motor power around the hips, the presence or absence of deformity, the action of the hips during gait with particular reference to the adductor-abductor components of hip function, and the stability of the hips. In this classification of function there were four grades, namely, *excellent, good, fair,* and *poor.*

With an *excellent* result the patient could walk without abnormally adducting the legs during locomotion, without scissoring, and without the lurch characteristic of a Trendelenburg gait. The hips were stable with good abductor power and at least 60 degrees of combined abduction (Figs. 2-C, 2-D, 3-B, and 3-C).

With a *good* result the patient had the same range of combined abduction but showed weakness of the hip abductors with a positive Trendelenburg test, and a lurching gait without crutches.

The result was rated as *fair* if the patient, in addition to these findings, also had combined abduction of the hips that was less than 60 degrees.

With a *poor* result the patient had markedly limited abduction of the hips, weakness of the abductor muscles, scissoring, and instability of the hip joints.

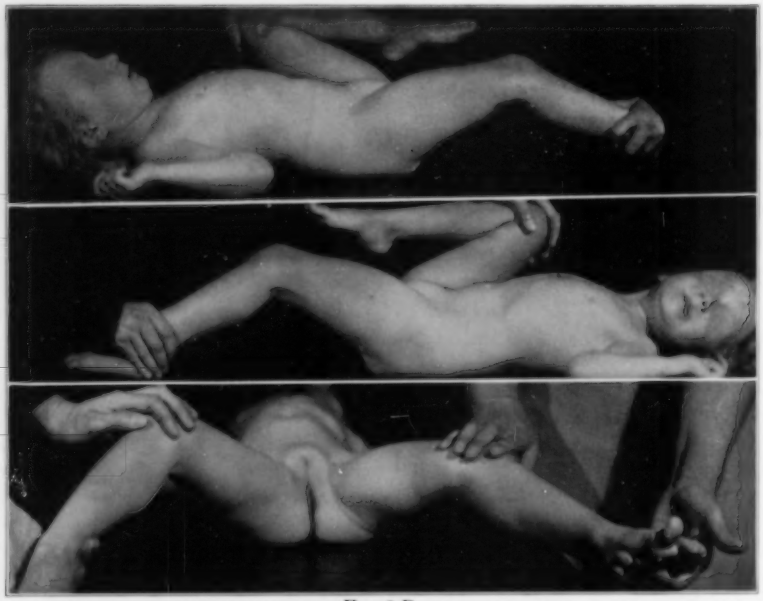


FIG. 2-B

Before operation. The patient in supine position. Note the hip flexion deformity and the restricted combined abduction.

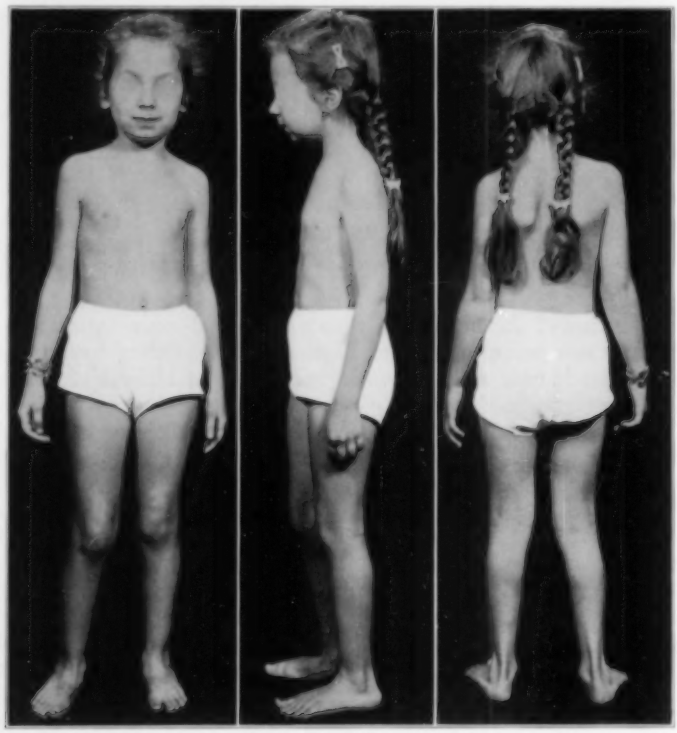


FIG. 2-C

After operation. The patient at the age of seven and a half years in January 1959, twenty-seven months after bilateral heel-cord lengthening, adductor myotomy, and obturator neurectomy.

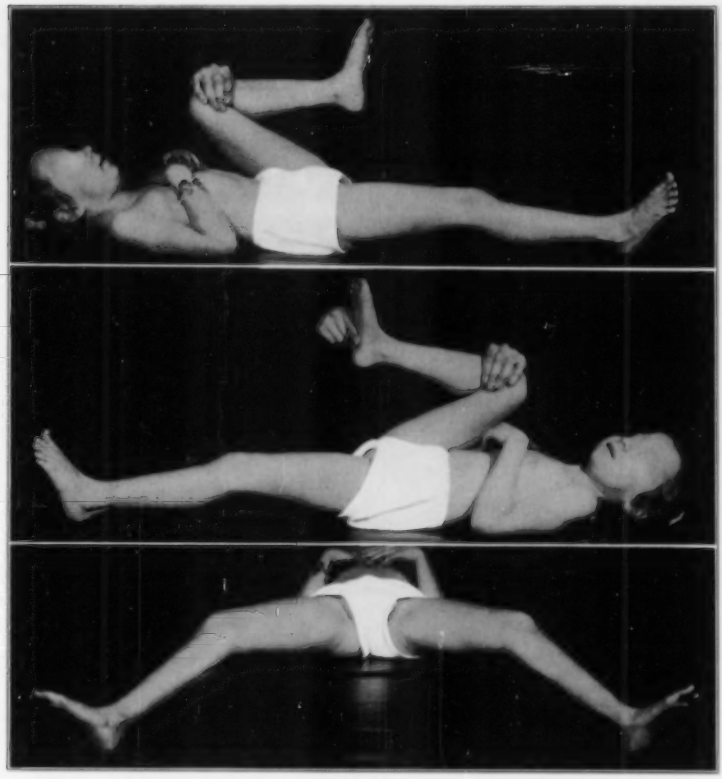


FIG. 2-D

After operation. The patient in supine position. Note the absence of hip flexion deformity and the wide hip abduction.

In considering the results obtained in these patients it was found desirable to relate each patient's general status to the result obtained by classifying all the patients studied into five categories based upon an assessment of their motor impairment. Five grades of motor impairment were selected ranging from motor status I, with minimal involvement, to motor status V, with severe involvement. This classification took into consideration the degree of spasticity, the amount of disturbance in coordination and balance, the severity of overflow responses, and the degree of motor control.

RESULTS

Of the seventy-four patients available for end-result study, thirty-eight had been originally classified in motor status V, twenty-seven in motor status IV, and nine in motor status III.

One hundred and forty-six adductor myotomies were performed on these seventy-four patients and could be evaluated (Table I). The over-all improvement after these procedures was graded as 4+ in fifty-six, 3+ in twenty-seven, 2+ in forty-four, and 1+ in eleven. Eight procedures in four patients who were in motor status V, with very severe involvement, resulted in *no* improvement.

The functional result following these adductor myotomies was rated as *excellent* in fifty-one, *good* in thirty-six, *fair* in twenty-nine, and *poor* in twenty-six procedures.

The number of patients on whom adductor myotomy was performed witnout obturator neurectomy was too small to compare as a group with those patients

on whom adductor myotomy was performed along with obturator neurectomy. In only two patients was bilateral obturator neurectomy performed without adductor myotomy. Adduction contracture was not a prominent factor in these patients. Two of these four procedures were rated as *fair*, and two as *excellent*.

In two of the patients with poor results, bilateral adductor myotomy had been performed primarily for nursing care. Although both patients were helped markedly as far as nursing care was concerned, the results were classified as *poor* because the patients did not maintain their correction, they were unable to stand or walk, and their hips remained dislocated.

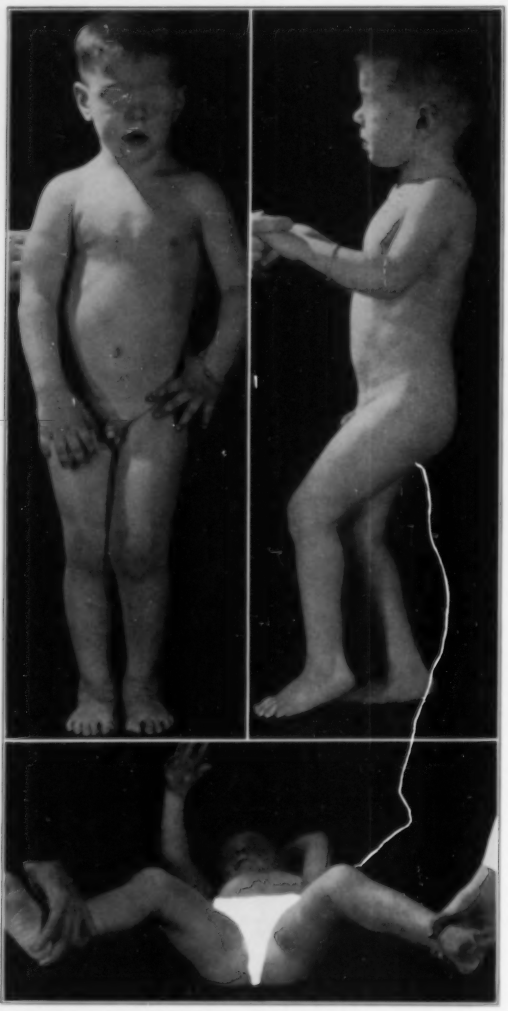


FIG. 3-A

Before operation. Spastic paralysis in P.G., age three years and nine months in December 1956. Note the restricted combined abduction.

Twenty-seven of the forty-eight patients who were not walking before operation were doing so at the follow-up examination. Ten patients were able to walk without assistance, and seventeen required crutches.

The ten patients who were able to walk without assistance after operation

were operated on at an average of six years and seven months (a range of two to thirteen years) and were examined for follow-up at an average age of fourteen years and nine months (a range of nine to twenty-six years). Sixty per cent of this group was moderately or severely retarded (intelligence quotients of 30 to 75).

The seventeen patients who required crutches for walking were operated on at an average age of five years and four months (a range of three to seven years) and were seen for follow-up examination at an average age of fifteen years and four months (a range of seven to twenty-four years). Eighty-two per cent of these patients were moderately to severely retarded.

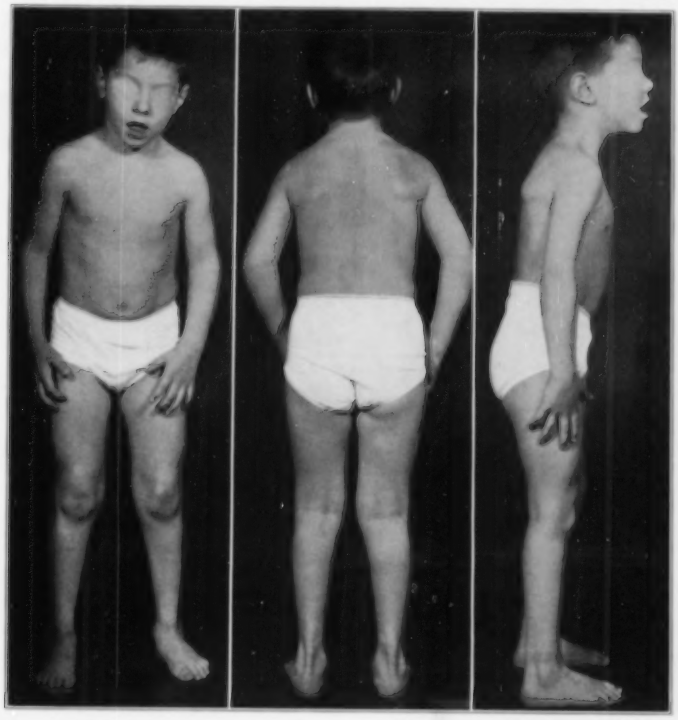


FIG. 3-B

The patient at the age of six years in March 1959, twenty-seven months after bilateral heel-cord lengthening, adductor myotomy, and anterior obturator neurectomy.

Nineteen patients, who were the most severely involved quadriplegics of motor status V with a very poor or absent sense of balance, were unable to walk with or without assistance at the follow-up examination, although two of them were able to get about with a walker. These nineteen patients were operated on at an average age of five and one-half years (a range of one year and nine months to twelve years and nine months) and were seen at follow-up examination at an average of twelve and one-half years (a range of three to twenty-four years). Eighty-five per cent of them were moderately or severely retarded. In eight patients, one or both hips were dislocated at the time of follow-up. No follow-up was possible in two patients.

Other Hip Deformities

Mild and moderate degrees of flexion deformity of the hip were benefitted

by adductor myotomy, especially if followed by appropriate stretching exercises (Fig. 3-C). Flexion deformity was otherwise unaffected by adductor myotomy. Positional internal rotation of the hips associated with scissoring and severe adduction contracture was uniformly improved by adductor myotomy and the postoperative regimen (Fig. 3-C), whereas fixed internal-rotation contracture often required other procedures. In no instance was an abduction contracture or deformity noted after operation or at the time of follow-up.

Hip Muscle Power

In 102 of the 146 hips evaluated, the abductor musculature at follow-up was rated as *fair* or better. Thirty abductors were rated as *poor*. In all of these, active muscular contraction of the abductors was noted, but the grading of the muscle was compromised by a limited range of hip motion. Ten of these thirty

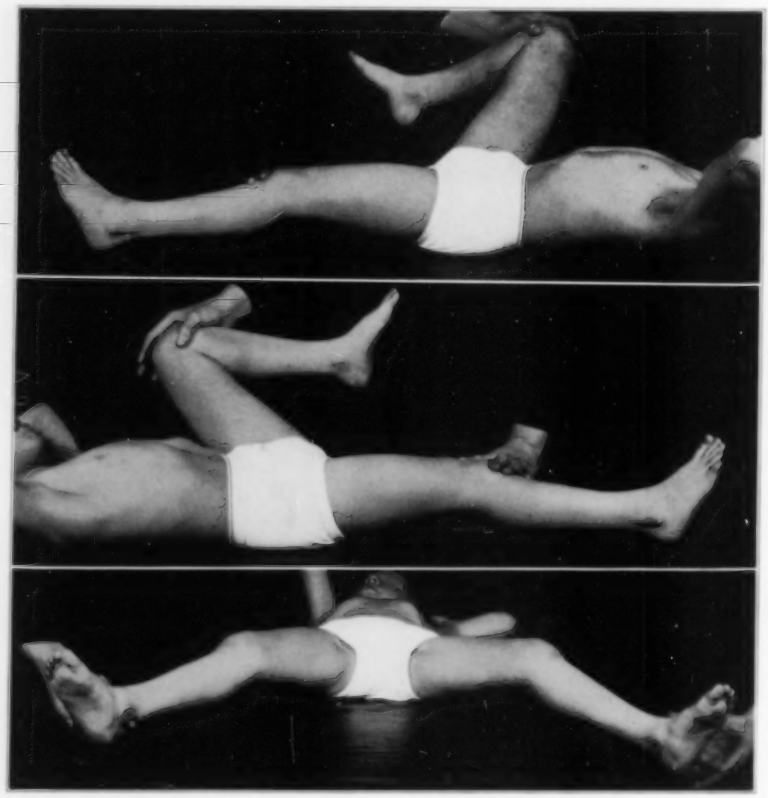


FIG. 3-C

After operation. The patient supine. Note the absence of hip flexion deformity and the wide combined abduction.

hips were dislocated at the time of follow-up examination, and in the remaining twenty there was residual adductor contracture. In fourteen of the hips the power of the abductors was not recorded. The adductors were rated as at least *fair* to *good* in all. It was not always possible to carry out a Trendelenburg test after operation because of poor balance in many of the quadriplegic patients. If the hips were stable, the balance of the patients was good, the follow-up adequate, and postoperative training of the abductors effective, good strength of the abductor musculature and a negative Trendelenburg were observed. In

thirty of the 146 hips, the Trendelenburg test was negative, and there was excellent motor control and function.

In no instance did adductor myotomy and obturator neurectomy result in weakness of the adductor musculature which might be responsible for difficulty in stance and gait.

All patients who showed evidence of adduction deformity of their thighs or scissoring after operation had residual spasticity and a recurrent contracture of the adductor muscles.

Subluxation and Dislocation of the Hip

In forty-two instances in which adductor myotomy and obturator neurectomy were performed, the hip on the side of the procedure was either subluxated or dislocated. Thirty-four of these hips showed subluxation, and eight showed dislocation.

Twenty-seven of the hips which were subluxated prior to operation were available for analysis. Of these, seventeen were normal on follow-up examination, five showed increasing subluxation, three were dislocated, and two were unchanged. In analyzing the cause of these poor results, it was found that almost all of these patients had had a minimum amount of care after operation, and a poor result might have been anticipated. Furthermore, in seven the procedure was done at an older age when there was already marked secondary disturbance in the development of the acetabulum.

There were eight hips in seven patients which showed frank dislocation prior to operation. Five of the dislocations in the older patients were very high, and reduction was not attempted. In four of these the adductor myotomy was performed solely to help in nursing care, and in the other it was done to improve gait as well. Of the three other hips, one was replaced by closed reduction after adductor myotomy at sixteen months of age and showed a femoral head in good position in a normal acetabulum at follow-up examination two years later. One patient had an open reduction of the hip at the age of ten and one-half years with resultant aseptic necrosis of the head of the femur, and one patient had no follow-up examination.

DISCUSSION

The management of adduction contracture of the hip in the spastic type of cerebral palsy has been the subject of as much discussion as have all other types of orthopaedic surgical procedures in cerebral palsy combined. It was stated [6] that procedures should not be performed before the end of the growth period and that surgery itself is of no value because of the high rate of recurrence of the deformity. The experience at the Children's Hospital from 1940 to 1956 with correction of adduction contracture of the hip in the spastic type of cerebral palsy has been as satisfactory as that of the correction of other deformities in this condition. Our experience has been that surgery adequately performed for proper indications and followed by very careful postoperative management yields good results (Figs. 2-C, 2-D, 3-B, and 3-C).

The prime indication for surgery was a severe scissoring associated with an adduction contracture of the hip that interfered with stance and gait in an individual with a fundamental sense of balance. On occasion, adductor myotomy combined with obturator neurectomy was performed before satisfactory evidence of balance could be demonstrated in a child whose hip was either dislocated or on the verge of dislocation.

Our findings would in no way substantiate the statement that surgery should not be performed in patients with cerebral palsy until their growth is completed [6]. All our patients in this series had their procedures before growth was completed. Seventy-two of the eighty-seven good and excellent results were in patients ten years of age or less, and, of these, thirty-seven were five years of age or less.

It was emphasized that adductor myotomy should not be performed in the presence of weak abductor muscles because the patient's existing ability to walk may be seriously imperiled. Under these circumstances it has been assumed that the patient requires the strength of the adductor muscles and the stability furnished by his scissors gait in order to walk [4,5]. In no instance during the period of this report were operations not performed because of weakness of the abductor muscles, and in no instance was the gait made worse by the procedure. In general, one can obtain evidences of the contractile power of the abductors prior to surgery if the examiner is persistent and tests the muscle under varying positions and under different circumstances. Even where contraction before operation was most difficult to obtain, the gluteus medius as a rule showed great improvement on the postoperative exercise regimen after the contracture of the adductors was relieved. It should be emphasized, however, that in the procedure which we performed, adductor power of reduced strength does remain.

TABLE I

RESULTS IN ADDUCTOR MYOTOMIES AND OBTURATOR NEURECTOMIES IN 175 TOTAL OPERATIONS ON EIGHTY-NINE PATIENTS

Operations	Total Operations	Follow-up Examination	Rating: Excellent	Good	Fair	Poor
Adductor myotomy and obturator neurectomy	157	135	49 (36%)	32 (24%)	29 (21%)	25 (19%)
Adductor myotomy	14	7	2	4	0	1
Obturator neurectomy	4	4	2	0	2	0
	175	146	53	36	31	26

IMPROVEMENT RATING IN COMBINED GROUP—175 OPERATIONS

No. of Examinations Not Done	No. of Follow-up Examinations	4+	3+	2+	1+	0
29	146	56 (38%)	27 (18%)	44 (30%)	11 (8%)	8 (6%)

Division of the adductors along with obturator neurectomy followed by adequate training was most effective in correcting the scissoring gait. If the patient was intelligent enough to work in an effective way and had a reasonable level of motor control, it was possible to develop increasing strength of the abductors and control of the hip so as to correct the waddling gait.

The adductor myotomy, furthermore, was most helpful in correcting the internal-rotation deformity in gait, provided that it was not severe. Flexion deformities of the hip as well were improved by the procedure. This was particularly true if active exercises of the gluteus maximus and stretching of the hip flexion contractures were a part of the exercise regimen after operation (Figs. 2-D and 3-C).

With a tendency to subluxation, early adductor myotomy with obturator neurectomy followed by immobilization in abduction was the procedure of choice, and tended to yield a good, stable hip. With frank dislocation which was recognized early this procedure was also effective. In such instances, exercises for a long period of time must be performed with the hip maintained in the abducted position. If the dislocation is not recognized until the individual is older, attempted reduction may be contra-indicated. Even in these patients, however, dislocated hips with fixed deformities may complicate nursing care and be painful. In such instances, adductor myotomy and obturator neurectomy may be indicated to relieve deformity and to simplify care.

The combination of adductor myotomy to correct the existing contracture

and weaken the muscles, combined with a neurectomy of the anterior branch of the obturator nerve to produce further weakness of the adductor group, has been the procedure of choice. We have not sectioned the posterior division of the obturator nerve since we wished to retain some strength of the adductors. We did not hesitate, however, to divide the anterior fibers of the adductor magnus to a greater or lesser extent, depending on the degree of contracture.

Even more important than the details of the procedure itself was the postoperative care. Those patients in whom the procedures were rated as having *good* or *excellent* results were, without exception, followed with good postoperative management. When the follow-up appeared adequate but the results were less than good, the patient presented a very severe retardation which undoubtedly compromised his ability to cooperate and to understand exercises and training. On the other hand, twenty-three of the twenty-six procedures rated as having *poor* results had very poor follow-up care and attention. The care of any patient obviously must be individualized, but, in general, the closer that the regimen followed the recommended plan of care, the more often was the result in the superior category.

In general, the principles of postoperative management involved wide abduction that was maintained until lengthening of the adductors was established permanently and the abductors reached a state of power which assured a satisfactory abductor-adductor reciprocal relationship. In the young patients, immobilization at night in bivalved casts with an abduction cross bar may be indicated for several years. These casts are initially worn continuously until gait-training is instituted and subsequently on a part-time basis, particularly at night. When immobilization is discontinued, the tendency to recurrence of the contracture must be looked for. If the balance between the adductors and the abductors has not been well established, the use of bivalved casts may again be necessary, particularly at night.

Combined with immobilization in abduction to obtain these objectives, a well outlined exercise regimen is essential. Particularly emphasized in the beginning are active guided contractions of the abductors. Later, active abduction exercises in the supine position, then against gravity, and finally in stance, are important. The transition into gait is ordinarily accomplished with crutches or, if the patient has severe involvement, with parallel bars. Crutches are used until a well balanced position of the hip during walking is established or until the maximum result has been obtained. In some cases crutches should be used during walking for any distances although they can be discarded for ordinary indoor activity. Individuals with poor balance may require crutches for this purpose indefinitely.

Exercises of a simple type to maintain the balance of the abductor-adductor mechanism may be necessary as a permanent part of the patient's daily activity. Release of a severe adductor contracture simplifies the problem of motor control in the extremity, and the exercise regimen contributes to improvement in other areas as well.

It is important that the patient have a mental status adequate for training. Twenty-one patients were considered to be of normal mentality, and sixty-four were definitely retarded.

Of the twenty-one patients who were evaluated as having normal intelligence, nineteen had follow-up examinations which permitted evaluation of the result. The results of thirty-six procedures on eighteen of the nineteen patients were evaluated as *good* to *excellent*, and the results of two procedures on the remaining patient were fair.

On the other hand, the results in the twenty-eight patients who were severely retarded (intelligence quotients ranging from 30 to 50) were less satisfactory. Follow-up examinations on twenty-four of these patients who had had forty-

seven procedures revealed good-to-excellent results after sixteen and fair-to-poor results after thirty-one of these operations.

The poor selection of cases was the major factor contributing to the poor results in this series. There were twenty-six procedures performed on sixteen patients who were rated as having *poor* results. These poor results could usually be explained on the basis of poor motor function, severe retardation, and very poor and inadequate follow-up with lack of cooperation on the part of the patient and family. Many of these patients had so little evidence of balance before operation that they probably should not have been operated on. If good results are to be obtained, the patient must have enough motor control and function to make the procedure worth while. Good sitting balance and reasonable standing balance as well are basic requirements for surgery.

In the previous report on surgical procedures in cerebral palsy from the Children's Hospital for 1925 to 1939 [3], the results of adductor myotomy were not so good as those in the present series. In the group reported earlier [3], the patients apparently were not immobilized for a long enough period of time in the abducted position after operation, physical-therapy measures were less emphasized, and the procedures were not extensive enough. The factors which contributed to the poor results in the first series were also responsible for the poor results in the present group of patients. Lessons learned from the first group were applied to the present series with much more successful results.

SUMMARY

Adduction contracture of the hip is the most common hip deformity in the spastic type of cerebral palsy. A high incidence of severe coxa valga, subluxation, and dislocation of the hip is associated with this contracture. In severe scissoring with adduction contractures of the hips, an adductor myotomy combined with anterior obturator neurectomy uniformly gives good results, provided that the patient's mental and motor status meet certain requirements. Adequate postoperative management and attention to certain minimum rules of care are major factors in the result. These patients must be followed over a long period of time for a maximum result to be obtained.

REFERENCES

1. Banks, H. H., and Green, W. T.: The Correction of Equinus Deformity in Cerebral Palsy. J. Bone and Joint Surg., **40-A**: 1359-1379, Dec. 1958.
2. Bradford, E. H.: The Surgical Treatment of Spastic Paralysis in Children. Trans. Am. Orthop. Assn., **3**: 7-14, 1890.
3. Green, W. T., and McDermott, L. J.: Operative Treatment of Cerebral Palsy of Spastic Type. J. Am. Med. Assn., **118**: 434-440, 1942.
4. Phelps, W. M.: Care and Treatment of Cerebral Palsies. J. Am. Med. Assn., **111**: 1-6, 1938.
5. Phelps, W. M.: Treatment of Cerebral Palsies. Clinics, **2**: 987-991, 1943.
6. Phelps, W. M.: Long-Term Results of Orthopaedic Surgery in Cerebral Palsy. J. Bone and Joint Surg., **39-A**: 53-59, Jan. 1957.

Pathological Fractures of the Spine
Etiology and Diagnosis

A Review of One Hundred and Five Cases *

BY JAMES A. NICHOLAS, M.D., PHILIP D. WILSON, M.D., AND ROBERT FREIBERGER, M.D., NEW YORK, N. Y.

From the Hospital for Special Surgery, New York

This is a report of a study of 105 patients admitted to the Hospital for Special Surgery between 1950 and 1958 because of back pain due to lack of bone mineral, with resultant vertebral compression. There was no history of adequate trauma or known malignant bone disease to explain such findings. All of the patients received a thorough step-by-step diagnostic study; the results were tabulated and analyzed and are presented in this report.

Among the more common causes of vertebral compression were osteoporosis, bone destruction from malignant disease, rheumatoid arthritis, and osteomalacia. Several important conclusions were drawn regarding: age at the onset of compression, number of vertebrae fractured, and indications for thorough diagnostic survey.

The word *demineralization* is used throughout this article in its roentgenographic meaning, which implies a diminished bone density owing to less than a normal amount of mineral [1]. Since it is the mineral substance that increases the density of bone over that of the surrounding soft tissues, it is only the amount of mineral and not the state of the bone matrix that can be evaluated by roentgenograms. The word *demineralization* as used here does not imply a physiological process whereby bone is removed from an intact bone matrix since evidence of such a process is lacking. Which terminology to use accurately for a description of too little bone is the subject of some debate [2].

SELECTION OF CASES

During the period of our study, 162 patients were admitted to the hospital with backache and roentgenographic findings of vertebral compression and demineralization of bone. Of these, fifty-seven were excluded from this study for the following reasons: twenty, because the force of injury was considered adequate to cause fracture of normal bone; eleven, who prior to admission were known to have malignant metastatic bone disease; and twenty-six, because of insufficient clinical, laboratory, or follow-up information. The data on the remaining 105 patients constitute the material presented in this report.

METHOD OF STUDY

Although many of the patients included in this report were studied extensively, it is not within the scope of this paper to present all of the information accumulated. Some of the results of the laboratory studies are not presented here since they are to be published elsewhere [4].

The data on which this report is based were obtained by the systematic, step-by-step diagnostic study outlined in Table I. The value of obtaining direct biopsy of a lesion or doing studies of the bone marrow is emphasized in Table II.

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 29, 1959.

TABLE I

DIAGNOSTIC STEPS EMPHASIZED IN INVESTIGATION

Medical History
Activity *versus* bedrest
Duration of symptoms, onset, and type of precipitating trauma
Menstrual history, ?pregnancy, ?breast feeding
Decrease in height and appearance of slouch
Gynecological, gastro-intestinal, or genito-urinary disease
Food intake, especially calcium in various forms
Medication, especially corticosteroids
Physical Examination
Posture
Multiple bone tenderness, specific localization in the spine
Motion
Height, weight
Motor or sensory changes
Evidence of systemic disease, especially in the gastro-intestinal tract
Laboratory Tests
Complete blood count, erythrocyte sedimentation rate, and urine (all patients)
Serum proteins:
Albumin-globulin ratio (65 patients)
Electrophoretic pattern (45 patients)
Serum calcium, phosphorus, and alkaline phosphatase (all patients)
24-hour urine calcium excretion on varying intake (47 patients)
Other special tests where indicated (stool fat content, renal function)
Radioisotopes, absorption loads
Roentgenograms
Chest (all patients)
Spine, skull, hands, or other indicated areas
Special studies, where indicated:
Intravenous pyelogram
Gastro-intestinal
Biopsy
Marrow, sternal, or iliac (48 patients)
Direct vertebral or other (25 patients)

RESULTS

Diagnoses

The data obtained in this study are recorded in Tables I, II, and III. Table III lists the diagnoses established. Although osteoporosis was the most common diagnosis made in patients over the age of fifty-five it was relatively uncommon in patients under this age. For this reason it was decided to analyze the results in two age groups—the seventy-eight patients over the age of fifty-five, and the twenty-seven patients who were under this age.

Patients over fifty-five years of age: Of the seventy-eight patients in this group, fourteen had primary or secondary malignant bone disease. Fifty of the sixty-four remaining patients were thought to have osteoporosis: forty-three, postmenopausal; four, senile; and three, idiopathic osteoporosis. When the term, *senile osteoporosis*, was used, it was to describe those patients in whom symptoms of demineralization developed more than thirty years after the menopause. This classification is retained because it has been used previously, although it has little, if any, scientific basis. Of the remaining fourteen patients, seven had hypercorticism induced by the use of corticosteroids in the treatment of rheumatoid arthritis, and four had osteomalacia secondary to malabsorption. Polycythemia vera, Gaucher's disease, and vertebral hemangioma or aneurysmal bone cyst were responsible for the fracture in the remaining three patients.

TABLE II

BIOPSY RESULTS

Procedure	No. of Patients	No. of Results Positive	Negative
Bone Marrow, Sternal or Iliac (total)	48	13	35
2 marrow examinations	9	1	8
3 marrow examinations	1		
4 marrow examinations	1	1	
Direct Biopsy (total)	25	10	15
Direct vertebral	6	3	3
Others	19	7	12

TABLE III

DIAGNOSES IN 105 PATIENTS WITH PATHOLOGICAL FRACTURES OF THE SPINE

Diagnosis	No. of Patients under Fifty-five Years	No. of Patients over Fifty-five Years	Total No. of Patients
Osteoporosis			
Postmenopausal	4	43	47
Senile		4	4
Idiopathic	1	3	4
	5	50	55
Malignant Bone Disease			
Metastatic cancer	7	7	14
Multiple myeloma	4	5	9
Leukemia		2	2
	11	14	25
Other Conditions			
Hypercorticism and rheumatoid arthritis	2	7	9
Osteomalacia (malabsorption)	2	4	6
Polycythemia vera	2	1	3
Hyperparathyroidism	2		2
Ankylosing spondylitis	1		1
Waldenström's macroglobulinemia	1		1
Gaucher's disease		1	1
Aneurysmal bone cyst		1	1
Myasthenia gravis	1		1
	11	14	25
Grand Total	27	78	105

Patients under fifty-five years of age: Of the twenty-seven patients in this group only five were diagnosed as having osteoporosis—four women and one man. In three of the women the menopause had occurred early in life, surgically induced in one at the age of twenty-nine years and of spontaneous occurrence in the other two at the age of thirty. The fourth woman, who was fifty-four years old at the time of this study, had had complete cessation of the menses at the age of fifty. Her severe bone demineralization and compression fracture, occurring only four years after menopause, were decidedly unusual. She was classified as having postmenopausal osteoporosis because laboratory evidence indicated a considerable amount of negative calcium balance and no evidence of other specific bone disease. The fifth patient was a man, thirty-eight years

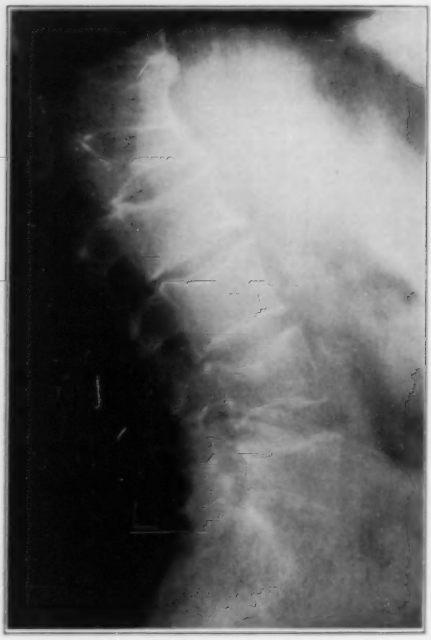


FIG. 1-A

FIG. 1-B

Case 22, L.K. Polycythemia vera.

old, with severe demineralization of the spine and of the bones of the extremities. His clinical and laboratory studies, including three bone biopsies, over a period of six years revealed only osteoporosis; he was thought to have idiopathic osteoporosis.

Eleven of these twenty-seven younger patients were found to have malignant bone disease: four had multiple myeloma, one had malignant lymphoma, and the others had metastatic carcinoma. In all eleven, pain due to the vertebral compression fracture and bone destruction was the presenting symptom preceding the discovery of the existence of the tumor. A number of unusual diseases caused the fractures in the remaining eleven patients in this group, but it should be noted that polycythemia vera was present in two.

Comparison of Findings and Relationship to Malignant Bone Disease

Because the data of patients with known malignant bone disease were eliminated from this study, and because postmenopausal osteoporosis was more frequent in the patient over fifty-five years of age, the findings indicated a higher percentage of malignant bone disease in the younger patients. A patient with demineralization and vertebral compression fractures who was over the age of fifty-five had, roughly, a 20 per cent chance of having malignant bone disease, whereas the chance of malignant bone disease being found in the patient under fifty-five with these symptoms was closer to 50 per cent.

Relationship of the Number of Fractures to the Diagnosis

Metastatic malignant bone disease was apt to be associated with a significantly smaller number of vertebral-body fractures than were the other conditions. Fourteen of sixteen patients with malignant bone disease had less than three compression fractures, whereas ten of the fifty-five patients with osteoporosis also had less than three compression fractures. Multiple myeloma produced a wide scattering in the number of vertebral-body fractures consistent

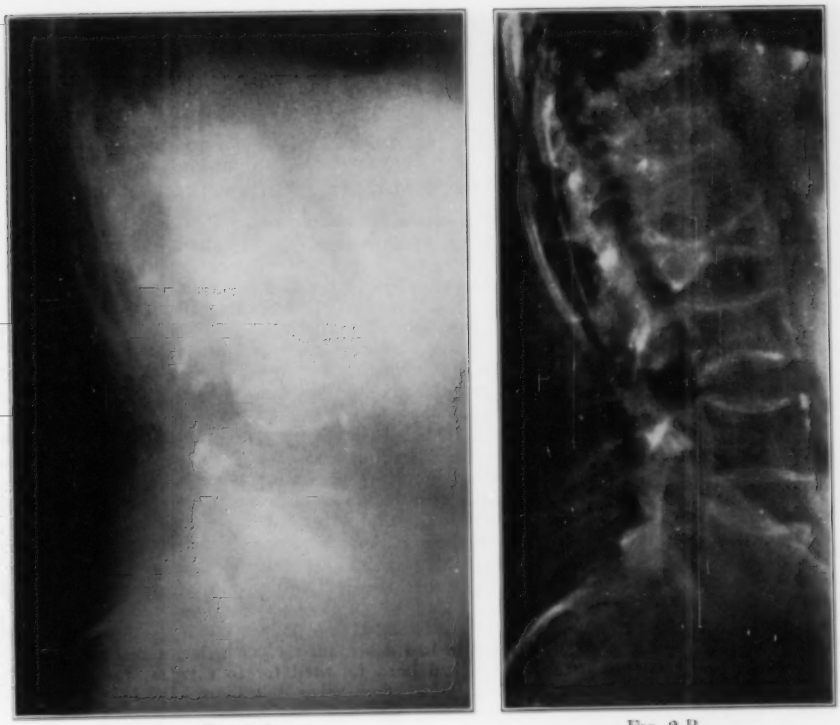


FIG. 2-A FIG. 2-B

Case 3, G.M. Waldenström's macroglobulinemia.

with the generalized process characteristic of this disease. With the exception of the patients with myeloma no patient with more than four compressed vertebrae in this group of cases had malignant bone disease. It should be emphasized, however, that in late metastatic disease any number of vertebrae may undergo destruction.

DIFFERENTIAL DIAGNOSIS—CASE REPORTS

The importance of establishing an accurate diagnosis in patients with pathological vertebral compression fractures cannot be overemphasized. Early diagnosis reduces deformity and indicates specific treatment in certain conditions. Ankylosing spondylitis, osteomalacia, polycythemia vera, hyperparathyroidism, tuberculosis, and, indeed, certain forms of malignant bone disease, are either curable or amenable to palliation.

Several interesting cases illustrate the wide variety of conditions other than osteoporosis and malignant bone disease, which must be thought of in the differential diagnosis, and illustrate the need for a thorough diagnostic survey.

CASE 22. L.K., a man, fifty-four years old, with known polycythemia vera for five years was admitted to the Hospital for Special Surgery because of severe back pain of five months' duration. Except for a peptic ulcer which had been discovered and treated effectively by diet ten years prior to admission, and a cholecystectomy for gall stones six years prior to admission, the patient had been in good health, with no restriction of activity or unusual intake of drugs. The polycythemia vera had been treated intermittently in the past; two phlebotomies had been performed and the patient had also received radioactive phosphorus.

On admission, physical examination showed the patient to be two inches shorter than what he considered his normal height. Roentgenograms revealed a round-back deformity with compression deformities of the twelfth thoracic vertebra and all of the lumbar vertebral bodies (Figs. 1-A and 1-B). Laboratory studies revealed a red-cell count of 10.5 million, a hemoglobin of 22.5 milligrams per 100 cubic centimeters, and a hematocrit of 70 per

cent. Red-cell volume was twice that of normal, and there was an increase in plasma volume of 25 per cent. Hyperplastic marrow with a disproportionate increase in megakaryocytes was found on sternal-marrow biopsy. Repeated serum phosphorus studies always showed a slightly elevated phosphorus, ranging about 5.0 to 5.5 milligrams per 100 cubic centimeters.

With additional treatment of the polycythemia vera, the patient's backache subsided, and no further shortening occurred.

Since there were three cases of polycythemia vera in this series, it should be seriously considered in the differential diagnosis of vertebral collapse.

CASE 3. G.M., a man, fifty-three years old, was admitted to the Hospital for Special Surgery for severe backache and for ileus, which had developed after lifting a moderately heavy object. The patient had had a similar attack five months prior to admission; he came to the hospital only after several chiropractic treatments had worsened the backache. The patient was known to have gall stones, and, in 1931, had been treated for syphilis with arsphenamine. In 1956, he had had a bunionectomy, after which a pulmonary infarct had developed. There was no history of dietary deficiency and no unusual intake of drugs.

On admission, physical examination revealed emphysema with a barrel chest, and roentgenograms showed vertebral compression fractures from the twelfth thoracic to the fourth lumbar vertebra with slight compression of the upper thoracic vertebral bodies (Figs. 2-A and 2-B). At the bases of both lungs there were peculiar infiltrations which had been present and unchanged for ten years; their significance was unknown. Pertinent laboratory data revealed the total serum protein to be 8.8 milligrams per 100 cubic centimeters, with 3.4 milligrams of albumin and 5.4 milligrams of globulin. Serum electrophoresis disclosed an elevated globulin fraction with diminished alpha 1 globulin and diminished albumin. Further studies by ultracentrifugation of the serum proteins revealed macroglobulins of high molecular weight. The sedimentation rate was elevated. All other findings were within normal limits, except for the results of the bone-marrow examination; these showed an increase in plasma cells and lymphocytes. It was thought that the complete findings suggested Waldenström's macroglobulinemia. Further questioning of the patient revealed that he had had two episodes of epistaxis and had recently had subcutaneous hemorrhages in his legs.

At the time of writing, the patient had been followed for four years; his height has declined from five feet, nine inches to five feet, five inches, but there have been no appreciable changes in the laboratory findings.

Waldenström's macroglobulinemia is characterized by the presence of globulins of high (near 1,000,000) molecular weights in abnormally high concentrations in the blood serum [3,6]. The diagnosis can be confirmed by special immunological methods. This rare disease must not be confused with malignant lymphoma, chronic lymphocytic leukemia, multiple myeloma, or cirrhosis of the liver—diseases in which macroglobulins have also been demonstrated.

CASE 90. B.L., a man, forty-five years old, was admitted to the Hospital for Special Surgery with the chief complaint of stiffness and pain in his back for five years. In an effort to have the cause determined, he had been admitted to three hospitals, and had consulted several doctors.

Complete diagnostic study failed to disclose any abnormalities in his roentgenograms or blood chemistry. His past medical history did disclose colitis for several years, a result of dysentery contracted in the South Pacific during World War II.

On his next-to-last admission to the hospital, roentgenograms showed compression fractures of six vertebrae and a loss of height of two inches (Figs. 3-A through 3-E). Comprehensive diagnostic search revealed excessive calcium excretion in the urine with persistent elevation of serum calcium. Studies of intravenous calcium tolerance, serum phosphorus, and renal tubular reabsorption of phosphorus were not conclusive for hyperparathyroidism. On several occasions, it was suggested to the patient that his neck be explored for parathyroid adenoma, but he refused. At no time was the erythrocyte sedimentation rate (Westergren) over twenty millimeters per hour. The patient's symptoms gradually disappeared, and the results of laboratory studies returned to within normal limits. Roentgenograms of the spine, one year later, disclosed typical irregular margins of the sacro-iliac joints and sclerosis typical of early ankylosing spondylitis.

Symptomatic relief resulting from steroid therapy was consistent with a diagnosis of ankylosing spondylitis, rather than with the previous diagnosis of idiopathic osteoporosis. For four years the diagnosis of this patient's condition had been idiopathic osteoporosis; it was only through persistent follow-up examination that the specific diagnosis of ankylosing spondylitis could be achieved. Corticosteroid treatment was then instituted, and marked improvement was obtained. The treatment, admittedly capable of inducing negative

FIG. 3-A

Figs. 3-A through 3-E: Case 90, B.L. Ankylosing spondylitis.
Fig. 3-A: March 30, 1954.

calcium balance, resulted in such relief that the patient insisted on the drug; it was gradually tapered off.

It was our impression that the combination of rheumatoid arthritis and corticosteroid treatment produced a particularly disabling and serious type of demineralization. Fractures of the spine were multiple, causing severe compression deformities. Osteoporosis due to disuse is undoubtedly an important contributing factor in such patients.

Of the six patients with osteomalacia in this series one had Milkman's syndrome, whereas the five others (of whom two were under fifty-five years of age) had laboratory findings consistent with osteomalacia. All had had severe gastro-intestinal disease at one time or another in their lives. It is not always possible to differentiate osteomalacia from osteoporosis by roentgenograms, except in the presence of Milkman's syndrome. It is important that a thorough attempt to differentiate these two diseases be made by laboratory studies and by biopsy. Diseases, such as osteomalacia, which involve a disturbance in mineral metabolism rather than in bone matrix, yield to specific therapy. Thus, the patient's condition can be markedly and often rapidly improved.

CASE 62. I.S., a woman, sixty-eight years old, had had back and bone pain for a number of years and, because of this, had voluntarily limited her activities. Before her admission to the Hospital for Special Surgery she had been seen by several physicians who had diagnosed her condition as osteo-arthritis and as reactive depression.

FIG. 3-B
March 20, 1957.

FIG. 3-C
January 31, 1958.

Physical examination disclosed an apparently well nourished woman who walked with a waddling gait. Medical history revealed that the patient had had a cholecystectomy when she was thirty-four years old and had been on a restricted diet since then, avoiding meat but drinking approximately one quart of milk a day. For three years prior to admission there had been a gradual and slight loss of weight. Roentgenograms revealed multiple symmetrical pseudofractures of the ribs with typical Looser's lines in the scapulae, the iliac bones, and the pubic rami. Similar narrow zones of demineralization were seen in both femoral necks, along with marked bilateral coxa vara. Examination of the spine disclosed vertebra plana of the vertebral bodies, as well as minimum anterior wedging of the thoracic vertebral bodies. This symmetrical compression of the softened vertebral bodies suggested a process of demineralization with retention of the more plastic non-brittle osteoid. Gastrointestinal roentgenograms were negative except for a small hiatus hernia. Laboratory studies revealed: alkaline phosphatase, 9.2 Bodansky units; phosphorus, 4.2 milligrams per 100 cubic centimeters; and calcium, 8.5 milligrams per 100 cubic centimeters. Blood counts were within normal limits, and the total protein was eight milligrams per 100 cubic centimeters. Urinary-calcium-excretion studies disclosed that with a 1,000-milligram intake, the output was 10 per cent; with a 250-milligram intake, however, the output was about 25 per cent. The patient improved rapidly and the Looser's lines disappeared after treatment with vitamin D and estrogenic sex hormones. However, the coxa vara and the pubic rami deformity persisted.

The final diagnosis in this patient was Milkman's disease. If the diagnosis had been made earlier, the severe hip deformities could have been prevented.

FIG. 3-D
January 31, 1958.

FIG. 3-E
March 20, 1957.

DISCUSSION

Osteoporosis of variable degree is usually present in the late decades of life, and spontaneous compression fractures are common at this time. The disease is rare before the age of fifty, but thereafter its incidence increases with each decade. Compression fracture in the earlier years should be regarded with a high index of suspicion. Even after severe trauma, the possibility of some underlying pathological process of bone should not be lost sight of. The diagnostic investigation in these instances should be thorough; they should include biopsy and, if necessary, should be repeated in six months. Biopsy is important in that it may establish some disease other than osteoporosis as the etiological factor in the collapse. We have found open biopsy preferable to aspiration biopsy with a needle, although aspiration biopsy in the hands of many investigators is adequate in lower areas of the spine.

Occasionally, no diagnosis can be made except osteoporosis. If there are only one or two vertebrae fractured, one should not accept this diagnosis without re-examination in six months and, if necessary, at intervals thereafter, until all other possibilities have been excluded.

One word of warning should be voiced concerning the roentgenographic interpretation of the degree of mineralization. It is difficult to diagnose demineralization accurately in the absence of compression. When there is diminished bone density with one or more pathological vertebral compression deformities—the criteria utilized in this report—demineralization is not difficult to recognize. The earlier roentgenographic criteria of bone demineralization of the spine include a generalized loss of density of bone; thinning of the vertebral end-plate; a relatively increased density of the thin end-plate when compared with the vertebral body; and the loss of the normal trabecular pattern, with increased

prominence of the vertical arrangement of bone trabeculae. Such findings, however, are rather unreliable since they cannot be measured and recorded accurately. Furthermore, their appearance varies with the technique of roentgenographic examination. Increased or decreased kilovoltage, amount and quality of the patient's soft tissues—fat or muscle—will greatly alter the over-all appearance of the bone density and the contrast on the roentgenogram. It must also be emphasized that even the slightest motion of the patient during exposure will blur, or even completely obliterate, the trabecular pattern of the bones on the roentgenogram. Yet, this motion may be difficult to detect either by observation of the patient during the exposure or by study of the roentgenogram.

There are many other rare diseases which may cause pathological compression fractures, but which were not encountered in this series. It must be remembered that the data we have presented represent those patients who have been admitted to a specialized hospital. Statistics derived from a similar study in a general hospital would be of interest.

A careful thorough clinical and laboratory diagnostic study should be performed in every patient under the age of fifty-five with collapsed vertebrae. This should include bone-marrow studies and direct biopsy of the lesion, if necessary. In older patients in whom there is a solitary collapse or persistent increasing deformity and pain, the same painstaking study is advised. Such a study requires four or five days in the hospital. A high sedimentation rate, an elevated serum or twenty-four-hour urine calcium level, an elevated alkaline phosphatase or mild elevation of the serum globulin require further study, particularly electrophoresis and dietary calcium control studies.

Any of these laboratory findings should cause one to redouble his efforts to make a definite diagnosis, utilizing biopsy of the affected area or other zone of demineralization or study of the bone marrow. If these are negative, skeletal survey and investigations of the gastro-intestinal and genito-urinary tracts are indicated, as well as re-examination in six months to a year, when the findings are inconclusive.

CONCLUSION

The records were reviewed of 162 patients who had been admitted to the Hospital for Special Surgery since 1950 with compression fractures of the spine and demineralization. Fifty-seven of the patients were excluded from this study because the diagnosis was known at the time of admission, adequate trauma to cause fracture had occurred, or the diagnostic study or follow-up had been inadequate.

In the remaining 105 patients, osteoporosis was thought to be responsible for the vertebral collapse in fifty-five patients, malignant bone disease in twenty-five, hypercorticism with rheumatoid arthritis in nine, and osteomalacia in six patients. Other conditions were responsible for vertebral collapse in ten patients.

The chances of a patient having primary or metastatic malignant bone disease were markedly increased if fewer than three vertebrae were collapsed and the patient was less than fifty-five years of age. In the younger-age group especially, a variety of unusual conditions other than osteoporosis produced the demineralization. In order to make a final diagnosis, complete and, often, repeated studies are necessary. It is important to establish an exact diagnosis since some conditions that produce vertebral collapse respond to specific treatment.

REFERENCES

1. Babaiantz, L.: The Osteoporoses. Radiol. Clin., **16**: 291-322, 1947.
2. Cooke, A. M.: Osteoporosis. Lancet, **1**: 877-882, 1955.
3. Hanlon, D. G.; Bayrd, E. D.; and Kearns, T. P.: Macroglobulinemia—Report of Four Cases. J. Am. Med. Assn., **167**: 1817-1821, 1958.
4. Nicholas, J. A.: The Effect of Low and High Calcium Intake on 24-Hour Urinary Calcium

Excretion in Patients with Demineralizing Bone Disease. Read at the Annual Meeting of the Orthopaedic Research Society, Chicago, Illinois, January 23, 1959.
5. SNAPPER, I.: Bone Diseases in Medical Practice. New York, Grune and Stratton, Inc., 1957.
6. WALDENSTRÖM, JAN: Pathological Globulins and Protein Synthesis. Exper. Med. and Surg., 12: 187-191, 1954.

DISCUSSION

DR. ROBERT D. RAY, CHICAGO, ILLINOIS: There is, I believe, general agreement among those who studied bone resorption, that demineralization does not occur *in vivo*. However, to find a substitute term is difficult. As you all know, Albright used the phrase *too little bone*. Recently, Bauer, Carlson, and Lindquist coined the term *osteopenia*. As long as we agree that demineralization does not occur, I am willing to leave the argument on semantics to somebody else.

There are four steps in the scientific approach to any problem. The first step is organization of one's knowledge. This involves accurate observation, the elimination of non-essentials, and the inclusion of all essentials in some kind of a classification. This study certainly illustrates this step. From such a study, one can conclude that there is a large group of people with too little bone, even when one eliminates those cases of metastatic carcinoma, multiple myeloma, and other defined diseases. These patients are in a negative calcium balance during the active stage of their disease; they have certain characteristic roentgenographic changes, and certain morphological changes on a histological level.

The next step in the scientific method involves framing a question, or defining a problem. With regard to the case in point, one might logically ask why these patients have too little bone.

As the third step—having framed one's question—one can develop a theory or a hypothesis to answer this question. If we grant that the amount of bone present in the skeleton at any time represents a balance between bone formation and bone resorption, there are several theoretical ways in which too little bone, or osteopenia, can develop.

The amount of bone equals the amount of bone formed, minus the amount of bone resorbed. Expressed in a different way, we can say that the amount of bone equals the rate of bone formation, times the interval of time, minus the rate of resorption, times time.

The first possibility is that one can have a failure of formation with a normal rate of resorption. This is the condition to which Albright and Reifenstein applied the term *osteoporosis*.

The next possibility is that we can have a normal rate of bone formation, but an increased, or accelerated, rate of resorption. This condition so far has not been named; no one has studied this particular condition, and, only recently, methods have made it possible to measure the rate of bone formation.

The third possibility is that we can have increased rate of bone formation and increased rate of resorption. These conditions are called osteodystrophy, or in some cases, osteitis.

And finally, we can have a failure of normal calcification of the matrix, or osteomalacia. Having framed the hypothesis that these are the possibilities for having too little bone, our final step in the scientific approach is experimental proof of this hypothesis. Today, with isotope techniques, we have the tools at hand. There are laboratories, including those at the Hospital for Special Surgery, the laboratory under Dr. Bauer in Sweden, and our own laboratory—to mention a few—that are engaged currently in this task.

Although scientific papers and exhibits are not ordinarily dedicated, I think that this study, which represents an untold amount of labor, should be dedicated to the memory of a child.

DISCUSSION

HETEROGENOUS AND AUTOGENOUS-TENDON TRANSPLANTS

(Continued from page 110)

fate of the fibrocyte. The ground substance could be studied in a similar manner by utilizing differential histochemical staining techniques. The antigenicity and fate of collagen is more difficult to ascertain; with present methods it is extremely difficult to obtain pure collagen—even by acid extraction and subsequent reconstitution. C^{14}-glycine-labeled tendons or tendon transfers performed on scorbutic animals might be useful in this regard.

As the ultrastructure and clinical properties of the collagen molecule are revealed, we, who, perhaps more than any other group of surgeons, are at the mercy of uncontrolled fibrous-tissue formation, have a grave responsibility to be searching continually for applications of new fundamental principles to the clinical problems of scar formation in the healing wounds of human hands.

In Vivo Osteocyte Death

BY HAROLD M. FROST, M.D., DETROIT, MICHIGAN

Varying numbers of empty osteocyte lacunae in decalcified, stained sections of bone have been observed by many workers. The inference is that these cells died and became autolyzed. Some observers [10,13] estimated the degree of cell death and related it to age and vascular disease. They noted that there are more empty lacunae in extra-Haversian bone (interstitial lamellae) than in Haversian systems. To date, interest in this phenomenon has been largely academic. Recent developments in our knowledge of the physiology of bone indicate that certain phenomena, for example, osteocyte death *in vivo*, are of clinical interest. Other developments include the demonstration of skeletal storage and release of electrolytes and buffer materials [1,2,3,4,5,7,11,12], and the presence, during life, of microscopic fatigue fractures in bone [6].

The data in this paper indicate that the incidence of osteocyte death in human bone is, in part at least, a function of age. The precision of the data is limited for reasons that will become obvious, but the results are noteworthy nonetheless in view of the high incidence of dead osteocytes found.

MATERIALS

All specimens were from human beings and were obtained from twenty-one post-mortem examinations and twenty-four surgical procedures. The bones studied were four radii, one ulna, two humeri, fourteen clavicles, twelve ribs, one vertebra, two ilia, four femora, eight tibiae, three fibulae, and two metatarsals. Only three of the patients from whom bones of the lower extremity were obtained had evidence of vascular insufficiency, in contrast to those studied by Jaffe and Pomeranz, and Sherman and Selakovich. The age range of the patients in the study was from infancy to eighty-four years. There were thirteen female and thirty-two male patients. Although specimens from more than 160 patients were examined, some material had to be rejected because of inadequate sampling, or because of local and systemic disease that affected the bone-cell counts. In this connection it has been observed that the incidence of dead bone cells appears to be increased in the bones of patients with severe cardiac disease and with Paget's disease, as well as in the bones of patients receiving chemotherapeutic agents in the treatment of malignant disease.

The case material was divided arbitrarily into the following age groups: zero to thirteen, sixteen to twenty-three, twenty-six to forty-four, forty-nine to sixty, sixty-two to sixty-eight, and seventy-one to eighty-four years.

TECHNIQUE

Fresh, undecalcified, hydrated bone sections twenty to forty micra thick were made and stained with basic fuchsin by methods published elsewhere [7,9]. These sections were mounted in Harleco synthetic resin for microscopic examination.

In completely mineralized bone this staining technique demonstrates only the cells and the walls of the spaces in bone: osteocyte lacunae, canaliculae, Haversian and Volkmann's canals, primitive longitudinal canals, and cracks [8,9]. Accordingly, cellular material in the osteocyte lacunae appears as very dense, red matter which occupies the lacunae. Absence of such material leaves lacunar spaces unstained. The density of stain on the lacunar walls varies within wide limits so that it is occasionally difficult with an objective of low numerical aperture to inspect the interior of a densely stained lacuna. However, if an objective with a numerical aperture of over 0.95 is used, only about 1 per cent of the lacunae can-

TABLE I

Case	Bone	Age (*Years*)	Sex	Empty Lacunae (*Per Cent*) Haversian Bone	Extra-Haversian Bone	Cancellous Bone
923783	Rib	0	M	1.5 (fetal bone)		
12430	Femur	2 da.	M	0	0	0
922804	Rib	2 mo.	M	0	0	
924040	Rib	2	F	2	10	
933265	Rib	5	M	9	10	
879301	Clavicle	13	M	5	29	
906377	Femur	16	M	8	20	
804330	Radius	17	M	2	22	
804330	Prox. phalanx	17	M	8	16	
924416	Rib	18	M	7	34	
907127	Humerus	21	M	1.4	19	
907127	Ulna	21	M	3	20	
907127	Radius	21	M	2	18	
927890	Rib	23	F	5	14	
933423	Rib	26	M	22	49	
929137	Rib	29	F	31	49	
913048	Rib	36	M	68	81	67
919123	Clavicle	38	?	29	59	
741771	Tibia P/3	38	F	24	63	
741771	Tibia M/3	38	F	20	60	
741771	Tibia D/3	38	F	25	65	
741771	Fibula	38	F	30	60	
853071	Clavicle	41	F	13	40	
928734	Rib	44	F	12	25	
889538	Metatarsal	49	M	9	35	
928459	Rib	51	F	23	63	
897487	Clavicle	55	F	17	79	35
918255	Radial head	58	M			23
927602	Rib	59	M	38	58	
426260	Clavicle	59	M	60	75	
916719	Clavicle	60	M	27	56	
890793	Clavicle	62	M	4.0	78	
R. T.	Vertebra	63	M			40
12396	Tibia	63	F	21	62	
920886	Tibia	63	M	26	78	59
911112	Humerus	64	M			48
911112	Radial head	64	M			50
922299	Clavicle	64	M	53	92	
919913	Clavicle	65	M	21	59	
G. I.	Ilium	65	M			28
922578	Femur	67	F	5	88	
922578	Tibia	67	F	4	55	
922578	Fibula	67	F	20	87	
922578	Metatarsal	67	F	44	82	
12177	Tibia	67	M	31	75	
12177	Fibula	67	M	41	72	
12178	Tibia	67	F	23	47	
A. B.	Ilium	67	M			40
892435	Clavicle	67	M	30	78	46
A. C.	Femur	68	M	56	87	
924525	Clavicle	71	M	8	68	
11818	Tibia	72	M	67	73	
027176	Clavicle	76	F	16	67	
010736	Clavicle	76	F	49	89	
918888	Clavicle	83	M	67	87	
693106	Tibia	84	M		60	60

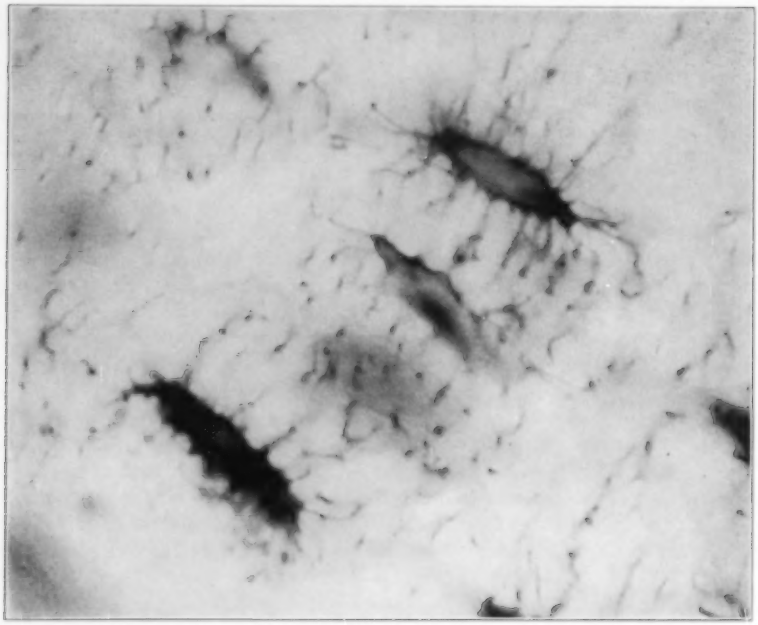


FIG. 1

Longitudinal section of a human clavicle. Three osteocytes are in focus; the one at 1 o'clock is empty, and the one at 7 o'clock contains stainable cellular material (basic fuchsin, × 750).

not be evaluated. Such lacunae are ignored in the counts. The bone from two patients with diabetes had to be excluded because a large fraction of the lacunae could not be evaluated.

A count was made of the number of empty lacunae and the number of those containing cellular material; the results were expressed as the percentage of the total lacunae that were empty. In each section, a minimum of 200 lacunae were counted, 100 in Haversian systems and 100 in the extra-Haversian* bone. The lacunae were counted in groups of five at twenty approximately equidistant intervals around the entire circumference of the bone. The fields selected were in the middle of the cortex. Since the incidence of cell death appeared to be related to age this procedure of selection was used in the hope of eliminating, as far as possible, any error caused by counting cells from either the newest or oldest bone in the cortex.

Since the sections were made by grinding the bone under water, some lacunae were opened at the ground surface and had debris forced into them which caused the affected lacunae to appear full of stained protoplasm. Inclusion of such open lacunae would have led to artificially low numbers of empty lacunae. For this reason, no such lacunae were counted. A microscopic objective with a numerical aperture over 0.9 was necessary to provide sufficient resolution and limited depth of field to avoid error in identifying the lacunae in the depth of the section. For this reason sections thinner than twenty micra were difficult to count.

There is a temptation to average the figures in Table I and II and thus arrive at an over-all average for a given bone. This would give an erroneous result, however, since the proportions of Haversian to extra-Haversian bone vary

* Extra-Haversian bone as used here refers in the main to interstitial lamellae. However, it includes all cortical bone except complete Haversian systems. In concentric systems, the innermost one is considered Haversian, whereas the outer ones are considered extra-Haversian. Distribution of empty lacunae, hardness, and mineral density would seem to justify this division.

TABLE II

Age Group (*Years*)	No. of Cases	Empty Lacunae (*Per Cent*) Haversian Bone	Extra-Haversian Bone	Cancellous Bone
0 to 13	6	3	8	
16 to 23	5	5	23	
26 to 44	7	28	52	
45 to 60	7	33	66	29
61 to 70	14	32	73	44
Over 71	6	41	74	

Combined Age Group (*Years*)	No. of Cases	Empty Lacunae (*Average Per Cent*) Haversian Bone	Extra-Haversian Bone
0 to 23	11	4	14
26 to 60	14	30	59
61 to 84	20	35	73

with the age of the patient and the location in the bone. In very young and very old persons, most of the skeleton (70 to 80 per cent) consists of extra-Haversian bone. In the intermediate ages, the ratio shifts to 30 per cent extra-Haversian [6]. In addition, areas such as the tibial crest normally contain a denser population of Haversian systems than the medial and lateral surfaces. It is for the latter reason that only complete cross sections could be included in the long-bone material.

Since it usually was impossible on a longitudinal section to be certain whether a given lacuna lies in Haversian or extra-Haversian bone, and since each longitudinal cut contained only part of one quadrant of the cortex, no counts on longitudinal sections were included. Such sections, however, revealed that the cellular content of a lacuna often lay in one end rather than in the middle of a lacuna. Because of this it must be assumed that in thin, decalcified sections some lacunae appeared to be spuriously empty (Fig. 1).

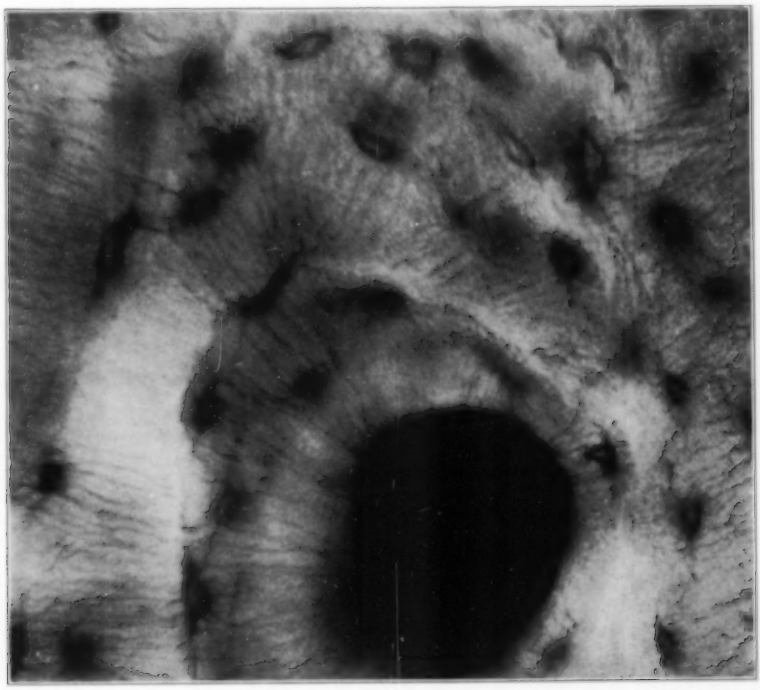


FIG. 2

Cross section of a human tibia. Most of a Haversian system is seen at the bottom of the figure, and most of the cells in the system contain stainable nuclear material. Most of the lacunae in the extra-Haversian bone are empty (basic fuchsin).

The fact that a lacuna contained stainable cellular material did not automatically mean that the osteocyte was alive when the specimen was obtained. Stainable cellular material has been seen in the lacunae of sequestra long after death of bone. In one instance such stainable material was identified in the lacunae of a sequestrum removed from a pool of pus three years after the sequestrum was first visualized by roentgenogram. Accordingly, the figures in Tables I and II for the percentage of dead bone are probably low.

An extensive statistical analysis of the data is not included since it is felt that the number of cases, number and variety of bones, and number of lacunae counted should be substantially increased before such analysis would be justified.

By doing repeated counts, it was found that if 200 cells per section were counted in twenty microscopic fields located at approximately equidistant points around the circumference of the bone, the maximum relative error was plus or minus 8 per cent. It is assumed, therefore, that the probable error in these counts is less than 8 per cent.

RESULTS

1. There is a steadily increasing incidence of empty lacunae with increasing age of the patient in the cases sampled.

2. In Haversian systems the average percentage of empty lacunae varied from less than 1 per cent at birth and in infancy to 40 per cent at age seventy.

3. In extra-Haversian bone the average percentage of empty lacunae varied from less than 1 per cent at birth and in infancy to 75 per cent at age seventy.

4. Up to the age of twenty-five, the percentage of empty lacunae in Haversian as opposed to extra-Haversian bone was in the approximate ratio of one to four. Beyond age twenty-five this ratio is about one to two, that is, there are half as many empty lacunae in Haversian bone as in extra-Haversian bone (Fig. 2).

5. In no case did the incidence of empty lacunae in Haversian bone equal or exceed that in extra-Haversian bone.

6. Osteocyte death is not limited to one bone, to one extremity, or to one type of bone.

7. There are wide ranges of individual variation in the percentage of empty lacunae. The range is appreciably greater in Haversian than in extra-Haversian bone at a given age. In cases from which more than one bone was obtained, the degree of cell death varied from bone to bone within narrow limits—less than the probable error of the counts. Insufficient material is at hand to evaluate the relation of the axial skeleton to the appendicular skeleton in this respect.

8. Osteocyte death is not a characteristic of a single disease. For example, in this study, the pathological conditions directly responsible for securing the specimen of bone in the age group of from fifty-one to sixty-eight years include gangrene, cirrhosis, myocardial infarct, sarcoma, traumatic amputation, fracture, coaretation of the aorta, mitral stenosis, metastatic malignancy, osteoporosis, sepsis, and fatal hemorrhage.

9. Cancellous bone appears, on the basis of four instances where cortical and cancellous bone from the same case were examined, to be involved less than extra-Haversian bone and more than Haversian bone.

SUMMARY

Counts of the percentage of empty osteocyte lacunae were done on fresh, undecalcified bone sections of specimens from forty-five human subjects ranging in age from new-born infancy to eighty-four years. The average figures from arbitrary age groups suggest that an increasing percentage of bone dies with increasing age. At seventy years, 45 per cent of Haversian bone and 75 per cent of extra-Haversian bone have empty lacunae.

REFERENCES

1. Bauer, G. C.: Metabolism of Bone Sodium in Rats Investigated with Na^{22}. Acta Physiol. Scandinavica, 31: 334-350, 1954.

2. Bergstrom, W. H., and Wallace, W. M.: Bone as a Sodium and Potassium Reservoir. J. Clin. Invest., **33**: 867-873, 1954.
3. Black, A. L.; Kleiber, M.; Smith, A. H.; and Ralston, N. P.: Mobility of Skeletal Phosphorus in a Mature Dairy Cow as Determined with Radioactive Phosphorus. Proc. Soc. Exper. Biol. and Med., **82**: 248-252, 1953.
4. Buchanan, D. L., and Nakao, Akira: Turnover of Bone Carbonate. J. Biol. Chem., **198**: 245-257, 1952.
5. Engfeldt, Bengt: Recent Observations on Bone Structure. J. Bone and Joint Surg., **40-A**: 698-706, June 1958.
6. Frost, H. M.: Papers read at the meetings of the Western New York Orthopaedic Association, Buffalo, 1956; the Orthopaedic Research Society, Chicago, 1957; and the New Jersey Orthopaedic Association, Paterson, 1957.
7. Frost, H. M.: Preparation of Thin Undecalcified Bone Sections by Rapid Manual Method. Stain Technol., **33**: 273-277, 1958.
8. Frost, H. M.: Some Observations on Bone Mineral in a Case of Vitamin D Resistant Rickets. Henry Ford Hosp. Med. Bull., **6**: 300-310, 1958.
9. Frost, H. M.: Staining of Fresh, Undecalcified Thin Bone Sections. Stain Technol., **34**: 135-146, 1959.
10. Jaffe, H. L., and Pomeranz, H. M.: Changes in the Bones of Extremities Amputated Because of Arterial Vascular Disease. Arch. Surg., **29**: 566-588, 1934.
11. James, A. H.; Brooks, L.; Edelman, I. S.; Olney, J. M.; and Moore, F. D.: Body Sodium and Potassium. I. Simultaneous Measurement of Exchangeable Sodium and Potassium in Man by Isotope Dilution. Metabolism, **3**: 313-323, 1954.
12. Levitt, M. F.; Turner, L. B.; Sweet, A. Y.; and Pandiri, Demetra: The Response of Bone, Connective Tissue, and Muscle to Acute Acidosis. J. Clin. Invest., **35**: 98-105, 1956.
13. Sherman, M. S., and Selakovich, W. G.: Bone Changes in Chronic Circulatory Insufficiency. A Histopathological Study. J. Bone and Joint Surg., **39-A**: 892-901, July 1957.

DISCUSSION

Acute Suppurative Arthritis of the Hip in the Neonatal Period

(Continued from page 30)

or by the parents themselves, with some type of antibiotic. Thus, with this overuse of antibiotics, initial infections were aborted before sufficient bone changes occurred for a diagnosis to have been made of incipient osteomyelitis, which, in many instances, might have been of the hip or the neck of the femur, with subsequent suppuration in the joint.

There has been a rising tide of criticism of the indiscriminate use of antibiotics, and justifiably so. However, this caution in renewed conservatism in the use of antibiotics—using them only where there is diagnostic indication—may result in an increase in the number of infections, such as those presented in Dr. Obletz's paper.

This makes it all the more important that we be alert and that we alert our colleagues, the pediatricians and the general practitioners, also to be ever alert to the possibility of septicemia and to infection that involves the hip joint. In the newborn infant, the period of grace between the onset of infection and destruction of the hip—the period in which it might be possible for the alert physician who made the correct diagnosis to do something about it—is very short.

Dr. Banks and I reported some cases years ago of regeneration of injured epiphyses, some of them from infection. But those epiphyses that were destroyed totally by the infection did not regenerate. Epiphyses that were partially destroyed, in many instances, showed a remarkable ability to regenerate. I am sure that some of you have wondered why the articular cartilage of some of the other joints seems to be so much more able to resist infection by pyogenic organisms than the hip joint. This has puzzled me for a long time. I think we can explain it on the basis of the circulation to the head of the femur and the ease with which that circulation is disrupted. Cartilage is nourished, to a considerable extent, particularly the articular cartilage, by synovial fluid itself. If the synovial fluid is turned into pus, the nutriment is missing. The distended joint which applies traction on the blood vessels supplying the neck of the femur and, indirectly, the cartilaginous femoral head, is sufficient to dislocate the hip. Certainly, that is sufficient to constrict the blood vessels considerably and to restrict the circulation to that femoral head. Perhaps, if it lasts twenty-four hours or longer, it could in itself produce a necrosis aside from the product of the infection attacking the articular structures. If an abscess of the bone is present, osteomyelitis in the neck of the femur, disrupting as it is, could perhaps produce weakness sufficient to cause a fracture and hence require, all the more, the application of a cast and immobilization to try to prevent final disaster. The head of that femur doesn't have much chance at all.

(Continued on page 150)

Micropetrosis

BY HAROLD M. FROST, M.D., DETROIT, MICHIGAN

In undecalcified, ground sections of human bone I have observed that the spaces of the canaliculae and osteocyte lacunae in some instances appear to be filled with mineralized tissue. This condition occurs during life, appears to be dependent on age, is not unusual, and may involve little or much of the skeleton. As reported elsewhere [3] I have coined the term *micropetrosis* to designate this condition.

Bone normally contains five types of spaces: the osteocyte lacunae, canaliculae, Haversian and Volkmann's canals, and primitive longitudinal canals [2]. In living bone these spaces are filled with cellular elements and extracellular fluid; they are physical discontinuities in the mass of mineralized matrix.

The observation that some of these spaces may become filled with mineralized tissue during life is not new. Heller-Steinberg, while working with undecalcified sections, noted occlusion of some canaliculae in rats. Although I have observed micropetrosis in slight degree in rodents, dogs, monkeys, and other animals, it is common only in man. Undecalcified sections are a prime requisite for the detection of micropetrosis.

MATERIALS AND METHODS

Material

I examined 109 bones from ninety-two patients—forty female and fifty-two male—ranging in age from new-born infancy to ninety years. Half of the material was obtained from surgical specimens, the other half from autopsies. Of the forty-two bones from the lower extremities only fifteen were from patients with vascular disease. A total of 392 sections was available for study.

Methods

Fresh, undecalcified, hydrated, unembedded bone sections fifteen to forty microns thick were made and stained with basic fuchsin, or prepared as water mounts or dry mounts, by techniques published elsewhere [4,6]. In studying the bone of the first twenty patients, all three types of mounts were used. The close correspondence of findings with the three types of mounts led to the selection of the fuchsin stain alone for the study of the remaining seventy-two patients.

The stained and dry-mounted sections were examined by brightfield microscopy; wet mounts were examined by phase and interference microscopy.

The microscopic appearance of basic fuchsin-stained undecalcified sections has been described elsewhere [5,6], but will be reviewed here.

Basic fuchsin molecules do not diffuse into normally mineralized* bone matrix at physiological temperatures, pH's, and pressures [5]. To demonstrate this the surfaces of the sections must be completely cleaned after staining. This was impossible with the techniques available to Heller-Steinberg. Because of the impermeability of normal fresh bone to fuchsin stains, only the walls of the spaces in the bone and the protoplasmic components in these spaces are stained. I believe that failure of the spaces to stain in the presence of adequate concentration of reagent is evidence of occlusion of the space by inorganic material. More direct evidence of this follows.

Dry mounts of undecalcified sections were made by warming each section to 40 to 45 degrees centigrade until all the free water evaporated and the protoplasm of the cells shrank to tiny masses. After warming, the spaces in the sec-

* No precise definition of normal mineralization exists. (This point is amplified in the paper entitled "Feathering" to be published in a subsequent issue of The Journal.)

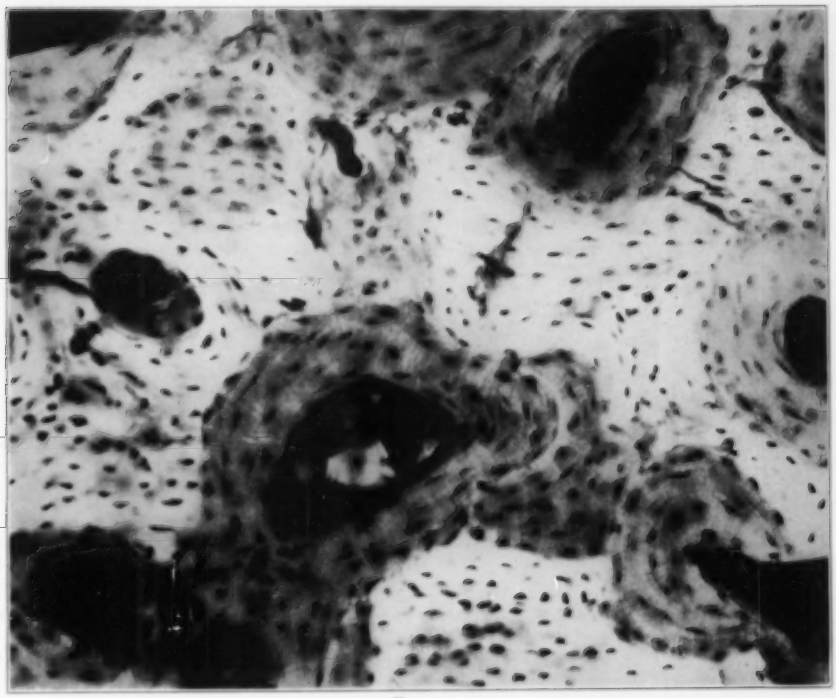


FIG. 1

Cross-section of the femur of a woman, sixty-seven years old. Haversian bone is stained and thus appears dark in the reproduction. Most of the extra-Haversian bone is micropetrotic, its canaliculae being filled with mineralized tissue. Some of the lacunae are similarly filled. The black lacunae in the micropetrotic areas are filled with air and unstained. On the whole, they are somewhat smaller in size than the lacunae in Haversian bone. This is presumed to be due to partial occlusion (basic fuchsin, × 150).

tion were shown to be filled with air. Because of the large difference in refractive index between bone and air—0.55—any aerated space was readily seen microscopically [1].

The sectioning technique used in this work involved grinding under water by hand. It was a simple matter to produce cracks in the section deliberately by increasing grinding speed and pressure. If bone were physically homogeneous, the cracks so produced would be expected to be randomly distributed, whereas if it were not homogeneous the cracks would be expected to develop first, and in greater numbers, in the more brittle portions. The location of these cracks, therefore, gives some indication of the physical characteristics of various parts of the bone.

Measurement

In order to determine the amount of micropetrosis in a given bone it is necessary to determine the proportion of the area of each section which is affected. If high precision was needed this would prove a formidable task. For the present purpose a method involving visual comparison was thought sufficient.

The method which was used consisted in selecting ten sections as standards. The degree of involvement in these sections ranged in easily discernible steps from the least to the most. The amount of micropetrosis in any one of these sections could be distinguished readily from that in any other by inspection with low magnification (30 ×). The percentages of the areas involved in these reference slides were measured with a Leitz integrating eyepiece micrometer to a precision better than 1 per cent. The remaining material was graded by comparison, using these slides as standards. The percentages of the areas involved in these

TABLE I*

Age Group (*Years*)	No. of Cases	Bones Used	No. of Sections	Average Grade (*Per cent*)	Range of Grade (*Per cent*)	Age Group Grade (*Per cent*)	Extremes
0 to 14	18	3 tibiae	20	1	0 to 1	1	0%, 16 cases
		1 fibula	7	0	0		
		1 metatarsal	1	0	0		
		15 ribs	50	1	0 to 1		
		1 clavicle	6	0	0		
15 to 55	25	2 radii	16	1.5	1 to 2	2.1	0% to 1%, 16 cases
		1 ulna	10	2	0		~ 5%, 5 cases
		1 humerus	10	2	0		
		9 clavicles	32	1.9	0 to 2		~ 10%, 1 case
		2 femora	12	2.5	3 to 5		
		6 tibiae	23	5	3 to 10		
		1 fibula	4	2	0		
		2 metatarsals	6	2	0		
		1 metacarpal	3	2	0		
56 to 69	33	2 radii	13	1	0	8	~ 2%, 10 cases
		1 humerus	10	1	0		~ 15%, 5 cases
		11 clavicles	28	2.3	1 to 15		~ 30%, 1 case
		2 ribs	6	10	5 to 15		~ 40%, 1 case
		1 ilium	2	1	0		
		2 femora	8	35	30 to 40		
		6 tibiae	34	17	5 to 30		
		5 fibulae	27	12	5 to 30		
		1 metatarsal	3	5	0		
70 to 90	16	3 clavicles	7	3	1 to 5	15	~ 2%, 2 cases
		1 rib	3	16	0		~ 15%, 10 cases
		1 ilium	4	40	0		~ 30%, 3 cases
		1 vertebra	1	24	0		~ 40%, 1 case
		2 femora	8	17	15 to 20		
		9 tibiae	23	15	1 to 40		
		1 fibula	2	20	0		
		1 metatarsal	2	5	0		

* Ten grades of involvement (Grades 1 to 10) were selected according to the percentage of the area involved: 0, 1, 2, 5, 10, 20, 40, 60, 80, and 100 per cent, respectively. These grades were selected on the basis of ten reference slides in which the differences in involvement could be recognized readily under low-power magnification (× 30). The percentage of the area involved in these reference slides was measured with a Leitz integrating micrometer and expressed in round numbers as indicated. In order to grade the bones studied, the percentage of the cross section involved in each bone was estimated by comparison with the reference slides. The eighth, ninth, and tenth grades (60 to 100 per cent) were sections obtained from the neighborhood of hip fractures of long duration. These grades do not appear in the table since they represent high degrees of micropetrosis seen only in pathological conditions. The column *Average Grade (Per cent)* lists the average grade of micropetrosis (expressed as per cent) of the indicated bones in the age group specified. In the next two columns to the right are recorded the range of grades for each bone and the average grade for each age group. The group average was determined from the grades of the cases in the age group and not from the grades of all bones studied in each group. The *Extremes* column records the number and percentage of involvement of the cases in each group with either less or more involvement than the average for the group.

slides were then rounded off to the values of the nearest reference group (Table I).

Because there was a marked variation in the amount of micropetrosis in the various quadrants of each cross section of bone, only complete cross sections of long bones were measured, the exception being twelve of the tibiae, in which only the anterior half of the bone was available. Since the amount of micropetrosis was found consistently to be maximum in the posteromedial quadrant of the tibia, the results obtained for the twelve tibiae reported here are assumed to be low. The average age of the twelve patients whose tibiae were studied was fifty-eight years.

Most of the sections were taken from the middle third of the diaphysis. Hence, the results obtained in this study do not reflect differences in the amount of micropetrosis at different levels of the bones studied. Early in this study cross sections from the upper, middle, and lower thirds of a number of tibiae and femora were compared. Serial sections from the same area varied less than 10 per cent in the amount of micropetrosis, whereas cross sections from the upper, middle, and lower thirds varied by as much as 100 per cent, the most marked differences being between the metaphysis and diaphysis. The amount of micropetrosis along the diaphysis varied little, and the changes were gradual. Hence, the level in the diaphysis from which the sections were taken did not appear to influence the results appreciably.

RESULTS

Microscopy

The most striking thing about micropetrotic bone was the absence of staining in the canaliculae in the stained preparations and the absence of air in these spaces in the dry mounts. Large variations in the amount of micropetrosis were found in different cases. In the lesser degrees of involvement the only canaliculae occluded were those just outside the cement lines at the peripheries of the Haversian systems, for the most part in the extra-Haversian bone (interstitial lamellae). As the degree of involvement increased, the extra-Haversian bone was observed to become progressively more affected, leaving the Haversian systems with their red-stained lacunae and canaliculae surrounded by colorless unstained bone. With still more marked involvement the Haversian systems were also affected. However, the innermost concentric lamellae of these systems were usually unaffected (Figs. 1 and 2).

Some of the lacunae in the micropetrotic bone were also found to be occluded to both the stain and air. However, in contrast to the canaliculae most of these appeared completely empty, with the result that they filled with air during the preparation of the section and were full of air in the final mounts. Considerable heating was needed to cause the mountant to fill these aerated lacunae, and they remained unstained.

Phase and interference microscopy of thin, wet mounts revealed that micropetrotic canaliculae and lacunae showed decreased contrast and increased optical-path length compared with unaffected canaliculae and lacunae in the same section. When the section was decalcified by flowing dilute acid under the cover slip all distinguishing features of micropetrosis disappeared, and the affected areas were in every way similar to the unaffected areas. This is additional evidence that the substance filling these spaces is mineral, at least in part.

Examination of the lacunae in micropetrotic bone in both stained and unstained preparations revealed that none of the lacunae appeared to contain cells.

The deliberate production of cracks during the grinding of sections from micropetrotic bone revealed that the cracks appear first in micropetrotic portions of the bone and outnumber those in Haversian systems by more than twenty to one (Fig. 3).

Grosser Features

The percentage of the area of a section that was micropetrotic varied markedly from quadrant to quadrant of the bone. In the tibia, as a rule, 1 per cent of the lateral surface and 20 to 30 per cent of the posteromedial aspect of the same section were affected. The location of maximum involvement was curiously standardized in the bones studied. At the intertrochanteric level of the femur maximum micropetrosis occurred in the calcar, whereas at mid-shaft it was in the posteromedial quadrant. The plantar quadrant of the metatarsals and the posterior borders of the fibula were similarly affected.

Investigation of multiple sections of many bones in the same patients sug-

FIG. 2

Cross-section of the tibia of a man, seventy years old. The Haversian canal is black. The inner member of this "double" osteon (Haversian system) appears dark because its canaliculae and lacunae are stained. The apparent diffuse staining of this osteon is due to the presence of large numbers of stained canaliculae and lacunae which are deep in the section and out of focus (basic fuchsin, × 400). The bone surrounding the central osteon is micropetrotic, almost all of its canaliculae being occluded with mineralized tissue. The arrows indicate occluded lacunae which are unstained. The black lacunae in the micropetrotic bone are black because they contain air, which was introduced during processing of the section.

gested that when one section of one bone shows considerable micropetrosis, other sections of other bones of the same patient will also show comparable involvement.

The data in Table I suggested that micropetrosis was related to age. No micropetrosis was found in the bones of new-born infants but in the patients over seventy years of age, micropetrosis affected an average of 15 per cent of the cross-sectional area*. Marked individual variations occurred, some elderly patients exhibiting little (1 per cent) and others marked (40 per cent) involvement. Of the three patients who had involvement amounting to 40 per cent, one had an iliac biopsy for evaluation of osteoporosis, one an amputation for vascular

* The percentage of area of a cross section affected was assumed to be equal to the percentage of volume of the diaphysis of the bone affected when—as in the present cases—sections from multiple levels of a given bone revealed the same percentage of area involved.

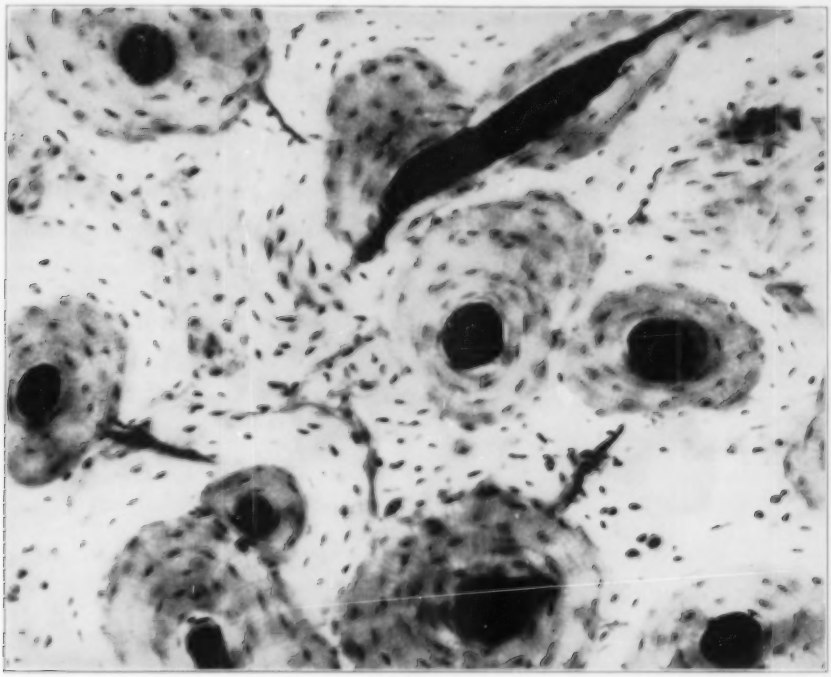


FIG. 3

Cracks produced during grinding (same case and stain as in Fig. 1). They occur only in micropetrotic bone. Several cracks can also be seen in Fig. 1.

disease, and one a subtrochanteric osteotomy to convert a fresh Pauwels Type III fracture of the femoral neck to a Type I.

Micropetrosis was found equally in both sexes in many bones and in diverse diseases. It occurred in cancellous as well as cortical bone.

DISCUSSION

Micropetrosis is a histological characteristic of bone demonstrable in ground sections, which appears to have significant implications with respect to the content and distribution of mineral. What factor or factors cause micropetrosis is unknown. Since maximum involvement in the femur, tibia, and fibula was observed in regions which might be assumed to be subjected to high concentrations of stress it is tempting to assume that the development of micropetrosis is related to strain.

The lesser involvement of the Haversian systems, even in the most severely affected parts of a bone, suggests that micropetrosis does not develop in these systems where better metabolic and diffusion processes are available.

The regularity with which a cement line appears to be the dividing line between the micropetrotic and other bone suggests that this structure plays some role in the development of the process.

The fact that cracks caused by grinding occur first and in much larger numbers in the micropetrotic bone suggests that this bone is more brittle.

The distribution of micropetrotic bone and its staining behavior suggest that it is relatively impermeable. Its age incidence likewise implies some relationship to the aging process.

SUMMARY

The term micropetrosis has been coined here to describe a bone condition in which the canaliculae and to a lesser extent the osteocyte lacunae are filled *in*

vivo with mineralized tissue. This condition was found primarily in extra-Haversian bone and appeared to be a characteristic of aging. Almost no micropetrosis exists at birth, whereas an average of 15 per cent of the volume of nineteen bones from sixteen patients over seventy was micropetrotic. In three cases 40 per cent of the volume was affected.

REFERENCES

1. Antonio, Ascenzi: Quantitative Researches on the Optical Properties of Human Bone. Nature, **163**: 604, 1949.
2. Bailey's Textbook of Histology. Ed. 10, pp. 115-121. Edited by Philip E. Smith. Baltimore, Williams and Wilkins Co., 1940.
3. Frost, H. M.: Papers read at the meetings of the Western New York Orthopaedic Association, Buffalo, 1956; the Orthopaedic Research Society, Chicago, 1957; and the New Jersey Orthopaedic Association, Paterson, 1957.
4. Frost, H. M.: Preparation of Thin Undecalcified Bone Sections by Rapid Manual Method. Stain Technol., **33**: 273-277, 1958.
5. Frost, H. M.: Some Observations on Bone Mineral in a Case of Vitamin D Resistant Rickets. Henry Ford Hosp. Med. Bull., **6**: 300-310, 1958.
6. Frost, H. M.: Staining of Fresh, Undecalcified Thin Bone Sections. Stain Technol., **34**: 135-146, 1959.
7. Heller-Steinberg, Minnie: Ground Substance, Bone Salt, and Cellular Activity in Bone Formation and Destruction. Am. J. Anat., **89**: 347-379, 1951.
8. Olsen, G. A.: Strength of Materials. Ed. 2. Englewood Cliffs, New Jersey, Prentice-Hall, Inc., 1956.

DISCUSSION

Acute Suppurative Arthritis of the Hip in the Neonatal Period

(Continued from page 143)

Dr. Carl Badgley, Ann Arbor, Michigan: I disagree with the cause of the loss of the head of the femur. First of all, I would like to say that these are neglected cases that Dr. Obletz is talking about. They are the evidence of the newer generation of doctors who have not seen suppurative hip disease as it was seen in the past. Dr. Obletz said that sixteen hips dislocated. First of all, when there is infection in the hip, it goes into a position of abduction and external rotation. This should have been recognized by the nurses or by anyone who took care of the infant. The dislocations are the result of ruptured capsules, and then the abduction deformity, and then the dislocation of the head. The absence of the head is not usually the result of Phemister's concept. It is the result of the dislocation, often producing an epiphyseal separation of the head from the neck of the femur; you will often find that these are displaced way posteriorly. The neck may again go back into the acetabulum. I feel that after observing 312 open operative procedures, which were reported in 1942, I can speak authoritatively and say that the loss of the head often is from sequestration. In my experience, if the head is separated, it dies.

Dr. Leo Mayer, New York, N. Y.: There is one phase of treatment which thus far has not been touched on: what to do in those instances where the femoral head is completely destroyed? I have had two such instances; both patients were approximately three months old at onset of the infection. I implanted the head of the fibula and then followed these patients for fifteen and eighteen years, respectively. The fibular head after transplant is not visible in roentgenograms; there is so little bone in it that you can hardly see it until after about five years. I noticed that in both of these patients the transplant is no longer adequate at about seven or eight years of age. The head begins to subluxate a little, showing that the fibula is not large enough to occupy the acetabulum and, therefore, I supplemented the first transplant by a transplant of the fibula of the other thigh. Both of these children walked fairly well at the age of fifteen and sixteen years, respectively. Unfortunately, symptoms of arthritis developed in one boy at about the age of eighteen and he is at present under treatment. I have had to perform two operations and drain the hip; the result, therefore, cannot be considered satisfactory. Despite antibiotics, it seems evident that some bacterial infection has remained latent in the femur over these years. I present these two cases, not as an example of what you should do, but as a request, or suggestion, as to other methods of replacing the femoral head which may be more effective than those I employed.

Plantar Dissection

An Operation to Release the Soft Tissues in Recurrent or Recalcitrant Talipes Equinovarus*

BY FREDERIC C. BOST, M.D.†, EDWIN R. SCHOTTSTAEDT, M.D., AND LOREN J. LARSEN, M.D., SAN FRANCISCO, CALIFORNIA

From the Shriners' Hospitals for Crippled Children, San Francisco Unit, San Francisco

Notwithstanding the conscientious and persistent employment of non-surgical measures for the correction of talipes equinovarus, there remain those deformities which recur after treatment and those which are recalcitrant or refractory to treatment.

At the San Francisco Unit of the Shriners' Hospitals for Crippled Children we found plaster-of-Paris, applied by the method of Kite, to be the most effective means for the non-surgical correction of talipes equinovarus. This method was used initially, regardless of the age of the patient, of the etiology of the deformity, or of the previous record of recurrences or failures after the use of this or any other method of treatment.

Despite repeated use of this treatment, there has been a considerable number of patients with recurrent or recalcitrant deformities. The treatment of these patients involves long and often repeated periods of hospitalization or care in the out-patient department. Throughout much of childhood these patients are left with feet that are not satisfactory, either functionally or cosmetically. In the past, these unsatisfactory feet—the result of conservative treatment—were treated by various operations. Triple arthrodesis was postponed, wherever possible, until the patient was in the later years of childhood.

The operation herein described was devised with the hope that an early correction of talipes equinovarus might be obtained, thus leading to the elimination of long hospitalization, repeated operations, and the eventual need for triple arthrodesis. The operation was first employed in 1946, and it is now believed that there has been sufficient experience with the results to present this report. No originality is claimed for this operation as it embodies the principles formerly used by many orthopaedic surgeons and especially those emphasized by Ober, Steindler, Garceau, and others. Furthermore, it does not differ greatly from the operation described by Brockman in 1930, an operation with which, unfortunately and inadvertently, we were unfamiliar when it was first utilized.

The etiology of talipes equinovarus is as yet unknown; there is still confusion concerning the pathological anatomy of this deformity. Its treatment, therefore, must remain on an empirical foundation. Nevertheless, experience would seem to indicate that treatment should concern the ligamentous tissues associated with the articulations of the navicular, the talus, and the calcaneus. However, the effect on the deformity of the intrinsic muscles of the sole of the foot and of such extrinsic muscles as the gastrocnemius-soleus, the tibialis posterior, and the tibialis anterior, cannot be wholly disregarded. The operation to be described was designed to effect the release of the aforementioned ligamentous tissues of the foot.

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 26, 1959.

† Deceased.

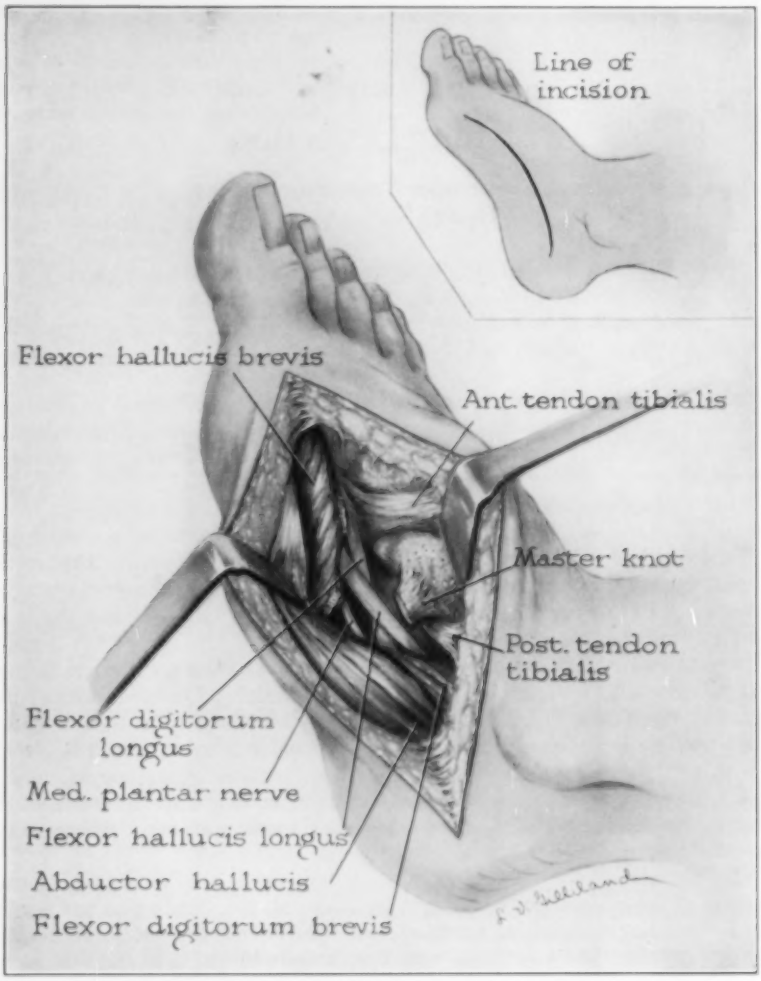


FIG. 1

DESCRIPTION OF THE OPERATION

The operation is performed with a tourniquet applied around the mid-thigh of the patient. The knee is flexed and a sandbag is placed beneath the opposite buttock to allow the foot to lie upon its outer side.

Incision

A medial incision, exactly as described by Henry for exposure of plantar structures, is used in this operation. The incision arches in a curvilinear manner from the medial tubercle of the calcaneus over the medial tubercle of the navicular and continues distally along the medial aspect of the internal cuneiform and the first metatarsal to descend at the middle of the first phalanx of the great toe. It is important to skirt the prominent medial border of the bones to facilitate the exposure of both the dorsal and the plantar surfaces of the tarsal joints (Fig. 1).

Development of the Exposure

The tendon of the abductor hallucis is detached from its insertion and the

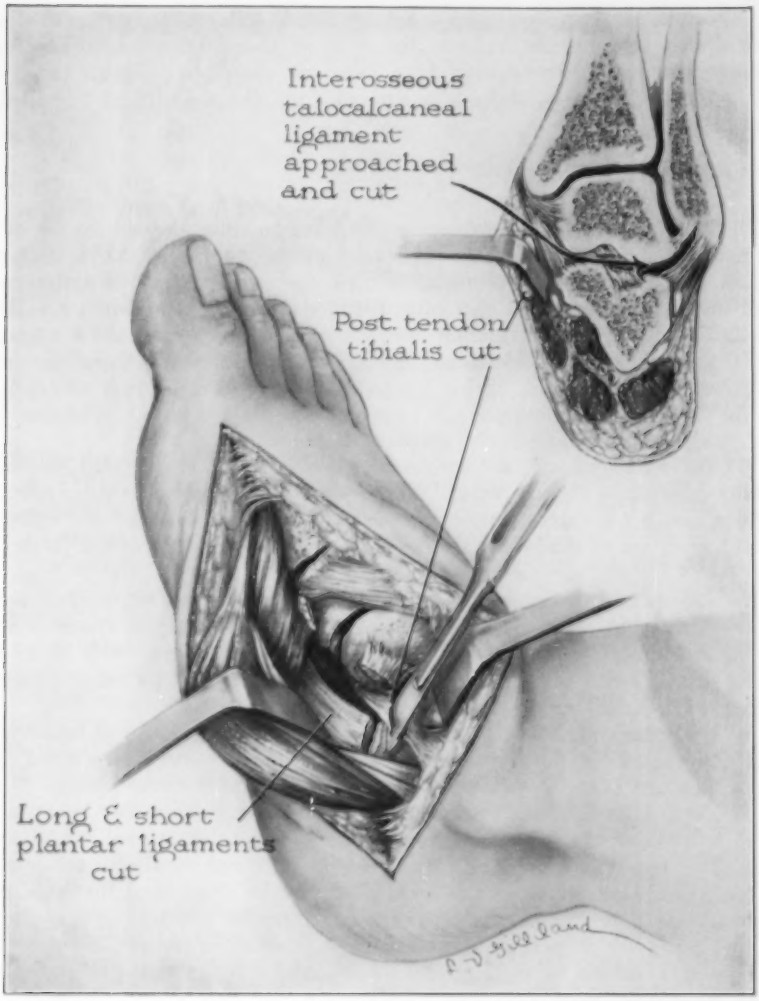


FIG. 2

entire muscle is reflected soleward from the dorsal fascial origin as far back as the medial tuberosity of the calcaneus. It is then possible to identify, although they are partially covered by a thin fascia, the neurovascular bundle of the tibial nerve and the posterior tibial artery with their divisions into lateral and medial plantar components, and the tendons of the flexor digitorum longus and the flexor hallucis longus. The latter are released by dividing the master knot of Henry beneath the tuberosity of the navicular and displaced plantarward. The origin of the flexor hallucis brevis is then divided, after which all of the intrinsic muscles of the plantar surface of the foot can, like the abductor hallucis and long flexor tendons, be reflected soleward, carrying the neurovascular bundle with them and protecting it from harm. The only structures not so protected are certain branches of the posterior malleolar and medial calcaneal arteries which will require ligation or coagulation.

The origins of the plantar intrinsic musculature, the ligamentous structures of the sole, and the deltoid ligament, together with the insertion of the posterior tibial tendon, are now thoroughly exposed to view (Fig. 1).

This beautifully planned approach of Henry follows along planes of anatomical cleavage and permits a perfect view of those structures to be divided in the correction of talipes equinovarus. The name chosen to designate this operation, *plantar dissection*, is in fact derived from the description provided by Henry.

The Correction of the Deformity

The operation is carried out in the following manner:

1. The insertion of the posterior tibial tendon into the tuberosity of the navicular, including its extensions forward to the cuneiform, as well as that portion which turns down soleward beneath the spring ligament and plantarward to the metatarsals, is carefully dissected from its attachment.

2. The origins of the plantar intrinsic muscles are divided just distal to the lateral and medial tuberosities of the calcaneus, allowing these muscles to fall soleward.

3. The long and short plantar ligaments are divided at the calcaneocuboid joint, together with the plantar capsule of this joint.

4. The ligaments of the talocalcaneonavicular joint are divided circumferentially around the entire proximal margin of the navicular. Particular attention is given to the plantar calcaneonavicular or spring ligament and the calcaneonavicular portion of the bifurcated ligament on the lateral side of the joint (Fig. 2).

5. The deltoid ligament is detached from the navicular, the medial margin of the calcaneonavicular, the sustentaculum tali, and, sometimes, from the inner side of the talus. This may be accomplished by inserting the knife blade next to and parallel with the talocalcaneal joint and sawing downward and upward to free the ligament as desired.

6. With the knife blade or a thin osteotome passed posteriorly between the talus and the calcaneus, the posterior talocalcaneal ligament is severed. This part of the operation is not, of course, performed under direct vision.

7. Next, attention is directed to the ligamentous structures between the talus and the calcaneus. The anterior capsule of the talocalcaneal joint is relatively thin, but it is divided. The interosseous ligament in the tarsal canal is divided, permitting access of the knife blade to the sinus tarsi for division of the ligaments that bind the talus to the calcaneus in this region (Fig. 2).

8. The naviculocuneiform-joint capsule is opened on its medial aspect and on the plantar aspect as far laterally as possible to free the navicular from all of the cuneiform bones.

9. The straplike insertion of the anterior tibial tendon is lifted (or detached if the tendon is to be transplanted at the same operation) from its insertion, and the cuneiform-first metatarsal ligament is divided medially and along the plantar aspect of the cuneiform-metatarsal joints. Care is taken to identify and to protect the insertion of the peroneus longus tendon. In some of the earlier operations the insertion of this peroneus longus tendon was also severed.

10. In those instances in which the equinus deformity is pronounced, a lengthening of the tendon of the gastrocnemius-soleus muscle may be performed at the same operation by curving the incision dorsally from the calcaneus. The tendon is ordinarily lengthened by the tri-cut method of Hoke. When the tendo achillis has been lengthened, it is moved aside to permit a direct exposure from the medioposterior aspect of the tibial talocalcaneal joint, the ligaments of which are divided under direct view.

11. If transplantation of the anterior tibial tendon is desired, this operation may also be done at this time.

After the release of the soft tissues, it is frequently possible to make a complete correction of the deformity by gentle manual manipulation. By this means the navicular is moved into its proper position overlying the head of the

talus, the calcaneus is brought downward and around the talus, and the fore part of the foot is moved into a straight position.

In the more rigid or fixed deformity, or where a secondary deformity of the bones exists, complete correction is often not possible at the time of the operation.

In either instance the foot is not held in a position of forced correction, for a correction which is too complete places too much strain on the soft tissues, the neurovascular structures, and the skin incision.

Closure of the Wound

Closure of the wound is effected simply by tacking the abductor hallucis muscle back up to the fascia from which it has been removed; a few interrupted sutures are used for this purpose. The subcutaneous layer and skin are then closed either separately with interrupted sutures or by vertical mattress sutures through the skin and subcutaneous tissue.

CARE IN THE POSTOPERATIVE PERIOD

After the closure of the wound, the foot is placed in a plaster cast in a position of gentle correction or, if this places the soft tissues under tension, in a position approximating its former state. The foot is held in the relaxed position until the wound has healed.

Although the line of the incision is an arched one, the majority of the wounds have healed by primary intention. Secondary healing after minor superficial sloughing of the line of incision has been seen in some instances, but rarely has a deep slough occurred along the line of incision. It should be emphasized, however, that the healing of the wound may require not two, but three or four, weeks' time, and that further correction of the deformity should be delayed until the healing has been completed.

After the healing of the wound, the gradual correction of the deformity has been effected by either repeated changes of the plaster cast or by the wedging method of Kite. The average time consumed in bringing about the desired correction of the deformity has been five and one-half months. In some instances nine to twelve months of plaster treatment have been necessary to effect correction. Thereafter, bracing has been used at night for varying periods of time, and regular or wedged shoes have been worn in the daytime. Those feet in which the deformity tended to recur have been managed by further periods of plaster-cast treatment or by subsequent operations, as will be noted in the report of the results of the operation of plantar dissection.

Indications for Operation

Plantar dissection has been applied to the treatment of talipes equinovarus, regardless of the etiology of the deformity. This report is restricted to the use of this operation for the correction of congenital talipes equinovarus or club-foot deformity (Figs. 3-A through 5-G). However, the operation has also been used effectively in talipes equinovarus accompanying other congenital disorders, such as spina bifida and arthrogryposis, as well as in deformities resulting from poliomyelitis, cerebral palsy, and other paralyses.

In the treatment of congenital talipes equinovarus, plantar dissection has been used when the deformity has recurred repeatedly after apparently satisfactory corrections had been achieved with plaster casts. The operation has also been employed in the treatment of recalcitrant deformities which simply were not corrected by plaster cast beyond a certain point.

We agree with those orthopaedists who believe that surgical treatment should not be used until the benefits of non-surgical treatment have been exhausted. We believe, however, that there is sufficient evidence to substantiate the fact that all deformities of congenital talipes equinovarus cannot be corrected by non-surgical treatment.

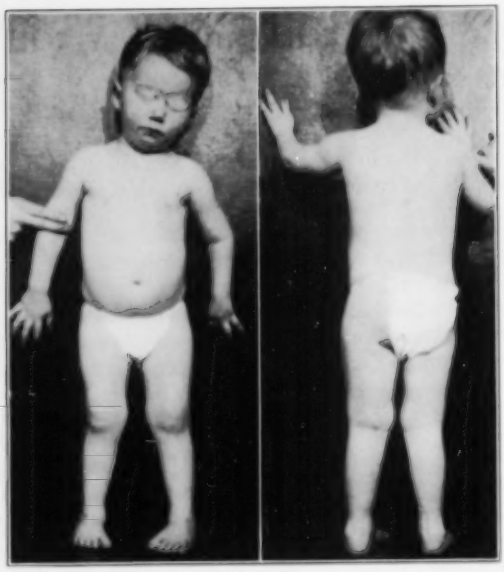


FIG. 3-A

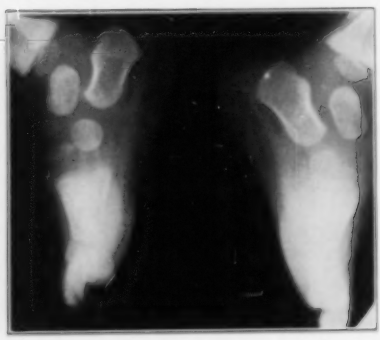


FIG. 3-B

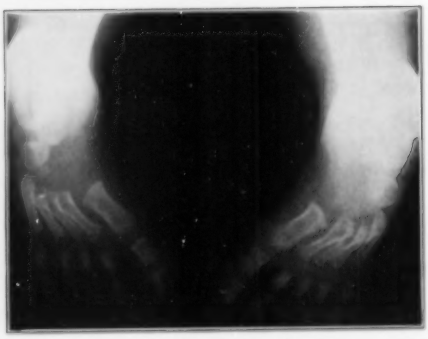


FIG. 3-C

Figs. 3-A through 3-C: P.M., born on May 23, 1943. Diagnosis: bilateral talipes equinovarus. Initial record: February 2, 1945. Denis Browne splints were worn from January 29, 1941, to October 21, 1944, after which a series of plaster-cast corrections was carried out until March 21, 1947. Surgery performed on December 3, 1947, consisted in bilateral plantar dissection, tendo achillis lengthening, and posterior capsulotomy. Follow-up plaster-cast correction was used until August 22, 1948.

All of the patients in the group reported here had been treated by plaster-of-Paris casts on more than one occasion. Many had received more or less continuous treatment over periods varying from six months to four years. Some, of course, presented themselves after long lapses of treatment; these patients were again subjected to courses of treatment by plaster-of-Paris casts, varying in duration from six months to three years. Many of the patients had previously had operations which included lengthening of the tendo achillis, incision of the posterior capsule of the ankle and subtalar joints, release or transplantation of tendons, minor osteotomies, and rotation osteotomy of the tibia.

STATISTICS CONCERNING PLANTAR DISSECTION

The Analysis of the Results

In the eight-year period from 1946 through 1953 there were 119 patients

FIG. 3-D

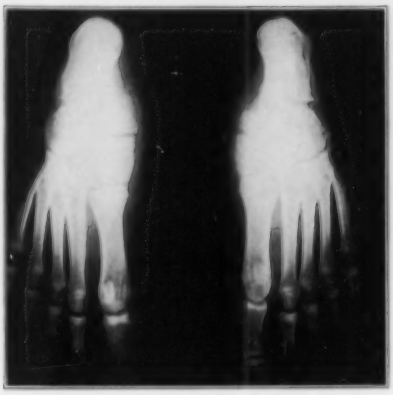


FIG. 3-E

Figs. 3-D through 3-G: Examination on January 21, 1957, showed the left foot to be overcorrected. Triple arthrodesis was performed on January 28, 1957.

FIG. 3-F

FIG. 3-G

with congenital talipes equinovarus who were treated by plantar dissection. Forty-nine of these had bilateral deformities, making a total of 168 operations.

Excluded from this number were a few older patients upon whom plantar dissection was done to correct the major deformity immediately before they received a triple arthrodesis.

Of these 119 patients, fifty-one (approximately 43 per cent) had reached the age of thirteen or more years at the time the assessment of the results was made for this report. Of the fifty-one patients, eight were thirteen years old, twenty-five were fourteen years old, thirteen were fifteen years old, and five were sixteen years old.

FIG. 4-A

FIG. 4-B

Figs. 4-A and 4-B: A.B., born on January 28, 1943. Diagnosis: left talipes equinovarus. Initial record: November 13, 1947. Plaster-cast correction was used for two years prior to surgery. Plantar dissection was performed on November 19, 1947, after which the patient was treated by plaster cast until July 29, 1948.

FIG. 4-C

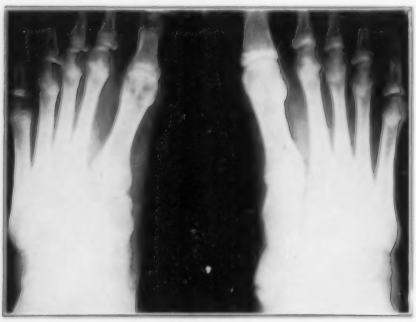


FIG. 4-D

Final record: May 3, 1956.

Although at these ages the ultimate outcome of the feet cannot be predicted with complete accuracy, it is believed that an analysis at these ages will give a satisfactory picture of the trend of the results from the operation.

The period of time after the operation at which the analysis in Table I was made ranged from two to twelve years.

The fifty-one patients whose results are reported in Table I had seventy operations for plantar dissection. Thus, this report covers approximately 42 per cent of the total operations (168) performed in the period from 1946 through 1953. The ages of these patients at the time the operations were performed ranged from three to thirteen years.

Thirty-seven, or slightly more than half of the seventy operations, included simultaneous lengthening of the gastrocnemius-soleus tendon. When an equinus deformity is present the lengthening of the tendo achillis is considered a part of the operation of plantar dissection.

Other operative procedures were carried out along with plantar dissection. In fourteen operations (20 per cent) the anterior tibial tendon was transferred to the cuboid (ten times) or to the outer cuneiform bone (four times). At the time of the initial operation on five feet, small wedges of bone were removed from the calcaneocuboid joint. In one foot the entire cuboid was removed to facilitate immediate correction of the foot deformity.

Subsequent to the plantar dissection, additional operations other than arthrodesis were necessary on sixteen feet to complete correction of the deformity. There were altogether thirty-six such procedures, some feet requiring more than one. These procedures included late transplantation of the anterior

TABLE I

PLANTAR DISSECTION (FIFTY-ONE PATIENTS)

AGE AT OPERATION AND YEARS OF OBSERVATION AFTER OPERATION

Age at Operation (*Years*)	No. of Patients	Length of Observation after Operation (*Years*)	No. of Patients
3	3	2	1 (14½ yrs. old)
4	9	3	1 (15 yrs. old)
5	8	4	4
6	8	5	5
7	7	6	8
8	6	7	7
9	3	8	5
10	4	9	6
11	0	10	8
12	2	11	5
13	1	12	1

TABLE II

THIRTY-SIX OPERATIONS SUBSEQUENT TO PLANTAR DISSECTION

Procedure	No. of Procedures
Transplantation, anterior tibial tendon	7
Original lengthening of Achilles tendon	5
Jones' extensor digitorum suspension	4
Interarticular arthrodesis, first toe	3
Replacement, anterior tibial tendon	3
Transplantation, flexor hallucis longus	3
Cole claw-foot operation	2
Osteotomy of all metatarsals	2
Osteotomy of first metatarsal	2
Removal of plantar exostosis	2
Tibiofibular osteotomy	2
Repeat lengthening of Achilles tendon	1

tibial tendon, replacement of the anterior tibial tendon where an overcorrection had occurred, lengthening of the tendo achillis where it was not originally lengthened, Jones' suspension of the metatarsal, and others, as listed in Table II. Plantar dissection was not repeated in any of the patients included in this series. This has not been true of the remaining, and as yet unreported, cases, where the repeated operation was carried out in several instances.

Assessment of Final Results

It is admittedly difficult to place an arbitrary assessment on the results of the treatment of deformities such as talipes equinovarus. The following values have been used:

Excellent: A normal, or nearly normal, gait, with dorsiflexion to at least 90 degrees or less, and with some motion in the tarsal joints. Normal motion has been experienced in some patients, but most of them have approximately 50 per cent of motion in these joints. The appearance of the foot was excellent, and there were no complaints of pain.

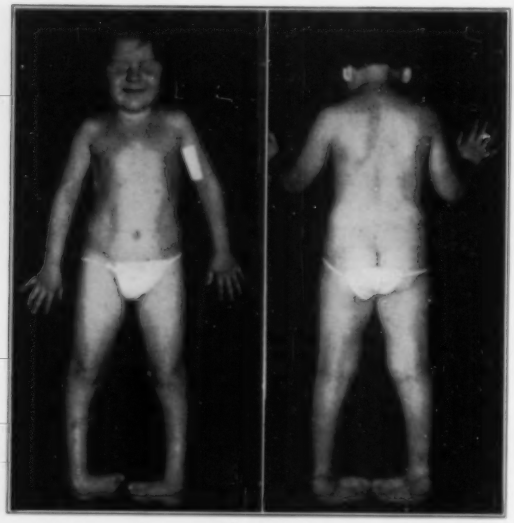


FIG. 5-A

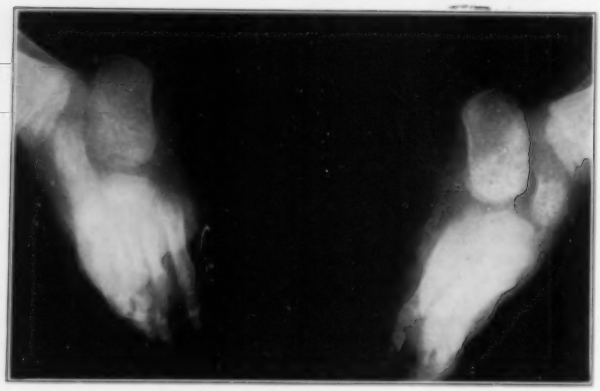


FIG. 5-B

FIG. 5-C

Figs. 5-A through 5-C: P.P., born on August 18, 1946. Initial record: November 15, 1952 (there had been no previous treatment). Bilateral plantar dissection was performed on November 19, 1952, and bilateral tendo achillis lengthening and bilateral posterior capsulotomy, on January 23, 1953. After the last procedure there was three and one-half months of plaster-cast correction.

FIG. 5-D

FIG. 5-E

Figs. 5-D through 5-G: Final record: February 20, 1958.

FIG. 5-F

FIG. 5-G

Good: Slight limitation of dorsiflexion, mild valgus position or overcorrection, and less motion in the subtalar and tarsal joints. The appearance of the foot was good, and there were no complaints of pain.

Fair: Overcorrection in valgus position to a moderate degree, some adduction of the fore part of the foot, considerable stiffness in the subtalar and mid-

TABLE III

RESULTS OF OPERATION

Procedure	No. of Procedure	Number of Results				Total Results	
		Excellent	Good	Fair	Poor	Satisfactory (*Per cent*)	Unsatisfactory (*Per cent*)
Plantar dissection	37	20	14	2	1	34	6
Plantar dissection with subsequent operations	16	4	7	2	3	30	30
					Totals	64	36
Triple arthrodesis	17				17		
Totals	70	24 (34 per cent)	21 (30 per cent)	4 (6 per cent)	21 (30 per cent)		

tarsal joints, and moderate disability. Symptoms of fatigue and of pain with use were present.

Poor: Marked valgus position or overcorrection or lack of correction with varus and adduction of the fore part of the foot, and stiffness and poor function of the foot, with complaints of pain.

Patients in the fair and poor classification might conceivably require triple arthrodesis at some future time. Those feet which did eventually require triple arthrodesis have been assessed as showing poor results after plantar dissection. The results of operation are shown in Table III.

TRIPLE ARTHRODESIS

Triple arthrodesis was used as the final treatment for the correction of the deformity in seventeen feet (24 per cent). This operation was performed from one to four years after the plantar dissection in seven instances, and from five to ten years after the plantar dissection in the remaining instances. Although these feet must be considered as failures in so far as the plantar dissection is concerned, they are, in fact, not bad as final results.

COMPLICATIONS

The immediate complications after operation were those associated with the healing of the wound. A few superficial sloughs and one deep slough occurred; none required either secondary closure or skin-grafting. Early embarrassment of the circulation was usually averted by avoiding a forceful correction of the deformity. Fortunately, there were no wound infections as a result of operation in this group of patients. Two patients complained of a sensitiveness in the scar or in the skin of the sole of the foot.

Late complications which accounted for poor results after this operation were stiffness of the subtalar, the tarsal, and, occasionally, the ankle joints; overcorrection of the deformity; overpull of the transplanted anterior tibial tendon; undercorrection of the contracture of the gastrocnemius-soleus tendon; and inability or failure to correct the other elements of the deformity, particularly the adduction of the metatarsals. A flexion contracture of the toes or an unusual drop of the metatarsals was rarely encountered.

DISCUSSION

Our experience in an institution devoted to the orthopaedic problems of childhood has pointed out the not infrequent occurrence of the recurrent or re-

calcitrant deformity of talipes equinovarus. To effect and maintain the correction of these deformities it is necessary to employ some surgical operation or operations. Plantar dissection, which some orthopaedists will condemn, is, admittedly, an extremely radical surgical means of achieving the desired correction.

It is clearly evident that the benefits of conservative management of the deformity should be exhausted before this operation, or any other, is employed. By the same token, a less radical operation should be used to obtain correction when possible. Equally evident is the fact that judicious care should be exercised in the selection of the foot deformities to which this operation is to be applied.

It is our opinion that plantar dissection serves a useful purpose in the management of the more difficult cases of talipes equinovarus. It is also our opinion that plantar dissection serves a useful purpose in the management of the intractable deformity of talipes equinovarus. This group includes those patients who have had prolonged treatment with no improvement or only very slight improvement, and those patients in the older-age group with neglected or recurrent club-foot showing severe deformity on initial examination. In the present series of deformities, the satisfactory results in 64 per cent of the operations have been gratifying. If the thirteen feet in which satisfactory results were obtained after triple arthrodesis are included, then 83 per cent of the seventy feet showed satisfactory results.

Furthermore, the operation is a satisfactory procedure to use in early childhood, when triple arthrodesis is undesirable. The beneficial effect of the early correction has been demonstrated repeatedly in operations on younger children. The operation has been employed on a number of occasions to correct recalcitrant deformity in the infant who is one to two years old. The results of the operations in infants and in some 200 other children are not included in this report, since these patients had not reached the age of thirteen years at the time this evaluation was made.

The correction after plantar dissection, even if incomplete, provides a useful functional result which persists through many years of childhood and permits final correction by arthrodesis with minimum removal of bone from the tarsus. The feet managed by this means maintain a more normal growth in length than do those treated by an arthrodesis in the early years of the preadolescent period.

As a result of the deformity, regardless of the type of treatment, many feet do not have normal flexibility. The loss of flexibility after plantar dissection is, of course, undesirable, but many feet so treated have retained motion amounting to 50 to 75 per cent of normal. Others have good correction of the deformity and good function despite 50 to 75 per cent, or even slightly more, restriction of motion. Improvement in surgical technique and improvement in management during the postoperative period have been effective in achieving better results in respect to the mobility of the foot. The age at which operation was performed does not appear to be an important factor in determining the amount of mobility obtained.

The analysis of the results has clearly demonstrated the importance of avoiding overcorrection of the deformity. Overcorrection has been an important cause of the poor results; these feet subsequently required triple arthrodesis. Overcorrection has been brought about by too forceful correction of the deformity, by unskillful technique in the application of plaster-of-Paris, and by overpull of a transplanted anterior tibial tendon.

In a few of the earlier operations, release of the peroneus longus tendon was employed to facilitate the correction of the adduction of the fore part of the foot; this sometimes resulted in a dorsal bunion. Release of the peroneus longus tendon has, therefore, been discontinued.

Increasing experience with this operation will permit more selective treatment of certain deformities. In this way, only that portion of the deformity remaining after previous plaster wedging or other modes of treatment need be attacked surgically. In some instances, this may lead to selective arthrotomy of the fore part of the foot or, again, of the hind part of the foot. In those feet with persistent tarsal varus (hind part of the foot), particular attention must be given to the release of the deltoid ligament and, especially, to the deep portion that inserts into the talus. In those feet with persistent deformity of only the fore part of the foot, it is unnecessary to sever this portion of the ligament.

Because of the complete exposure of the insertion of the anterior tibial tendon obtained in this procedure, there is a great temptation to transplant this tendon in association with the plantar dissection. The decision regarding the need for immediate transplantation of the anterior tibial tendon is difficult to make with accuracy. We have usually elected transplantation when the patient showed associated adduction of the fore part of the foot during walking. This adducting movement of the foot during walking gives the impression of an overacting tibialis anticus. This is in contradistinction to a foot whose fore part shows merely a static or fixed varus position. It is usually wiser to transplant the tendon to the outer cuneiform than to the cuboid bone (Figs. 3-A through 5-G).

SUMMARY

Plantar dissection, a radical surgical attack on the deformity of talipes equinovarus, has been described, and the results obtained by operation on seventy feet of fifty-one patients have been reported.

The indication for this operation is recurrent or recalcitrant talipes equinovarus. Of the seventy procedures performed, forty-five (64 per cent) resulted in satisfactory correction of the deformity, and the outcome in twenty-five (36 per cent) was unsatisfactory. Of these twenty-five operations, seventeen included triple arthrodesis (70 per cent of the total unsatisfactory results).

Some of the technical details leading to an overcorrection of the deformity have been emphasized, and our opinion regarding plantar dissection has been included in the discussion.

DISCUSSION

Dr. George J. Garceau, Indianapolis, Indiana: Recurrence of deformity and false or inadequate correction of deformity in club-foot is a problem that always faces us. There are numerous reasons why a foot may resist correction, regardless of how we treat it. When the joints of the foot are not normal, the muscles which move these joints function poorly and thereby cause return of the deformity; muscle imbalance will cause recurrence of deformity in a completely corrected club-foot. The ligaments of the medial side of the foot may be so resistant to stretching that correction is impossible and the tendo achillis may resist all efforts at correction. Therefore, one must always be alert to detect false correction and thus perform soft-tissue surgery before secondary changes occur in the bones and joints.

If the false correction, or incomplete correction, is allowed to persist the talonavicular joint remains partially dislocated, the subtalar-joint changes (changes in the joint) prevent derotation of the calcaneus, and the tibiotalar joint becomes flattened.

The operation described by the authors is apparently designed to prevent these secondary changes. I have not performed a thorough dissection as described, but I have performed at least partial dissection on the medial side of the foot. The unsatisfactory results were due to inadequate dissection and releases, especially in the older-age group.

Of the fifty-one patients reported by the authors, only twenty were under five years of age. In the series not reported, many were younger and in these, further surgery may not be required; that is, triple arthrodesis and other combinations of other operations may not be necessary. The authors transferred, at the time of plantar dissection, the insertion of the anterior tibial muscle-tendon laterally in fourteen instances. I would be reluctant to combine these two operations unless the peroneal muscles were dysplastic or very weak. In three of the fourteen feet, it was necessary to return the insertion of the anterotibial tendon to the medial side of the foot. My experience parallels that. However, in seven patients it appeared necessary to transpose the anterotibial tendon insertion laterally subsequent to the plantar

(Continued on page 176)

Sulphate Metabolism in Cartilage

II. S^{35}-Sulphate Uptake and Total Sulphate in Cartilage Slices * †

BY ROSE ROBYNS COELHO, PH.D., AND O. DONALD CHRISMAN, M.D.,
NEW HAVEN, CONNECTICUT

From the Department of Orthopaedic Surgery, School of Medicine, Yale University, New Haven

In this study, the uptake and loss of sulphate by chondroitin sulphuric acid in cartilage slices was investigated under a variety of experimental conditions. Although much previous work has been done on the incorporation of sulphate labeled with radioactive sulphur (S^{35}) into the ground substance of cartilage slices, the variables that govern the total sulphate content of cartilage *in vitro* have not been studied to our knowledge. We hoped that this information might help in the understanding of some of the changes in degenerated cartilage, the partial loss of metachromasia, the fibrillation, and the loss of elasticity which accompany the vicious circle of traumatic arthritis familiar to all orthopaedic surgeons.

The uptake of S^{35} sulphate *in vitro* by strips of cartilage and tumor tissue was first described by Layton [14] with the assistance of one of us (O.D.C.) in 1949. This technique has since become a useful tool for the investigation of ground substances. In 1951 Dziewiatkowski isolated radioactive chondroitin sulphate from the articular cartilage of rats given intraperitoneal injection of $Na_2S^{35}O_4$ and demonstrated the importance of cellular activity in sulphate metabolism by autoradiography in 1952. Boström and Månsson and Boyd and Neuman showed that this pickup of S^{35} sulphate was not by adsorption or diffusion, but by incorporation of the tracer into the chondroitin-sulphate molecule, and that various enzyme inhibitors strongly influence this reaction.

In this paper we are concerned with the effect of iodoacetic acid and sodium cyanide on the uptake of sulphate labeled with radioactive sulphur by slices incubated in Krebs-Ringer's solution. The effect of incubation itself and of the enzyme inhibitors on the total sulphate content of cartilage was also investigated in an effort to gain some understanding of the dynamic equilibrium which governs the integrity of the chondromucoid complex.

METHODS

Solutions

Krebs-Ringer's solution, buffered to pH 7.5 to 7.6 with Sørensen's phosphate buffer, was used for all experiments. To it, glucose (one gram per liter) and disodic sulphate (Na_2SO_4) (150 milligrams per liter) were added.

S^{35}-labeled Krebs-Ringer's solution was prepared by the addition of approximately 150 microcuries of carrier-free $Na_2S^{35}O_4$ to one liter of the Krebs-Ringer's solution. Sterilization of all solutions was performed by autoclaving for twenty minutes, and the buffer was sterilized separately. Following sterilization all solutions were exposed to oxygen bubbled through sterile tubing for thirty minutes.

Experimental Procedure

Cartilage was obtained from the ribs of puppies, two to six months old, using nembutal anesthesia. The cartilage was freed from surrounding connective tissue

* Read at the Annual Meeting of the Orthopaedic Research Society, New York City, January 31, 1958.

† Aided by a grant from the Medical Fluid Research Fund of the Yale School of Medicine and by the American Cancer Society.

and most of its perichondrium and was stored in Krebs Ringer's solution at 37 degrees centigrade until the end of the operative procedure. The cartilage was sliced in the Krebs-Ringer solution, and three to five slices per sample, each slice about one millimeter thick, were placed in incubation tubes with S^{35}-labeled Krebs-Ringer's solution. In some of the experiments, various enzyme inhibitors, including sodium cyanide (NaCN) and iodoacetate (each 1×10^{-3} moles per liter), were added to the incubation medium for comparison. In others, samples were heated for one hour at 45 degrees centigrade. The tubes were immediately placed in the incubator at 37 degrees centigrade and shaken throughout the experiment. Solutions were changed at three-hour intervals. Sterile conditions, as proved by negative cultures, were strictly maintained throughout the procedure. After the number of hours specified for each experiment, the slices were carefully blotted on filter paper, weighed, and transferred to Kjeldahl flasks containing three milliliters of concentrated nitric acid. In the preliminary work, the weighing was done before as well as after incubation, but this was found to be inadvisable. After digestion at the boiling point, the cartilage-strip residues were transferred quantitatively to twenty-five-milliliter volumetric flasks and diluted to the mark. Aliquots of this were used for total sulphur determination and S^{35} counting.

In the final experiments, S^{35} was omitted from the incubation medium, but it was supplied by the intraperitoneal injection of eight millicuries of $Na_2S^{35}O_4$ into puppies four days before the cartilage slices were obtained. Thereafter, the slices were used as just described, with and without sodium cyanide (NaCN) and iodoacetic acid (IAA). Aliquots of the pooled incubation media were also counted.

Analytical Procedures

One-milliliter aliquots from the diluted nitric acid digest were transferred to stainless-steel planchets, dried, and counted with a windowless counter (Tracerlab scaler). From the counting data on these aliquots and the counting data obtained from the S^{35} Krebs-Ringer's solution used for incubation, the amount of S^{35} taken up by the cartilage slices was calculated. Using this, the total sulphate pickup from the incubation medium (expressed in our table as milligrams of radioactive sulphur per 100 grams of cartilage) was calculated.

Fister's method for determining the total sulphur content was used on aliquots of the diluted nitric acid (HNO_3) digest with the following modifications:

1. The initial deproteinization with 10 per cent trichloracetic acid was omitted as being unnecessary;

2. Since the initial digest was relatively acid, the pH had to be carefully adjusted to 7.0 before the precipitation of phosphates with anhydrous aluminum chloride ($AlCl_3$);

3. The excess $AlCl_3$, eliminated with ammonium hydroxide (NH_4OH) gives a flocculent precipitate which can be separated more easily by centrifugation after storage in the deep freeze.

Except for the sodium cyanide (NaCN) experiment, reported in Figure 2, six to ten samples were incubated in the same medium.

All results are expressed in terms of milligrams of sulphate sulphur per 100 grams of cartilage. Sulphur in forms other than sulphate sulphur occurs in very small amounts in cartilage [12].

RESULTS

Weight Loss of Cartilage Slices

Great care in handling the cartilage slices and in maintaining their temperature at 37 to 38 degrees centigrade throughout all dissection and slicing procedures was essential. Preliminary experiments have shown that significant loss of weight occurred upon incubation in Krebs-Ringer's solution if these precautions just mentioned were not observed. For instance, cartilage sliced and weighed prior to incubation would lose up to 45 per cent of its initial weight in six hours, unless all

TABLE I

INCORPORATION OF S^{35} SULPHATE AND LOSS OF TOTAL SULPHATE BY COSTAL-CARTILAGE SLICES FROM A PUPPY TWO MONTHS OLD *

Hours of Incubation in S^{35}-Labeled Krebs-Ringer's Solution	Total Sulphate Content of Cartilage Slices (*Milligrams of sulphate sulphur per 100 grams of cartilage*)	Total Sulphate Loss (*Milligrams of sulphate sulphur per 100 grams of cartilage*)	Percentage of Sulphate Lost, Referred to 0-Time Value	$S^{35}O_4$ Uptake (*Milligrams of sulphate incorporated from S^{35}-labeled solution per 100 grams of cartilage*)
0	330.0	0.	0	0
3	286.0	44.0	13	3.30
6	270.0	60.0	18	3.55
9	253.0	71.0	21	3.80
12	270.0	60.0	18	4.42
18	234.0	96.0	29	4.97
30	237.0	93.0	28	5.46

* Each value is the average of four separate cartilage samples, each analyzed for sulphur in quadruplicate.

TABLE II

COMPARISON BETWEEN TOTAL SULPHATE CONTENT OF COSTAL-CARTILAGE SLICES FROM EACH OF SEVEN PUPPIES, TWO TO EIGHT MONTHS OF AGE, BEFORE AND AFTER INCUBATION IN KREBS-RINGER'S SOLUTION AT 37 DEGREES CENTIGRADE *

Experiment No.	Sulphate Content of Cartilage Slices Immediately after Slicing in Krebs-Ringer's Solution (*Milligrams of sulphate sulphur per 100 grams of cartilage*)	Sulphate Content after Twelve Hours of Incubation in Krebs-Ringer's Solution at 37 Degrees Centigrade (*Milligrams of sulphate sulphur per 100 grams of cartilage*)	Percentage of Loss of Total Sulphate
1	330	270	18
2	387	307	21
3	534	430	28
4	366	292	21
5	451	351	22
6	493	420	15
7	412	349	15
Average			20 ± 3

* Each value is the average amount determined in five to ten samples of cartilage, each analyzed in quadruplicate.

procedures were performed quickly and without allowing the slices to cool. To reduce handling of the slices, weighing was performed after incubation. Variations in weight associated with slight changes in pH have been observed by Blix and Snellman.

Sulphate Concentration of Cartilage Slices

Loss of sulphate was observed in all experiments upon incubation of the slices in Krebs-Ringer's solution, despite careful handling and proper temperature control. In Table I the time course of this phenomenon is shown. In Table II is given the total sulphate-sulphur content of cartilage slices incubated for twelve hours in Krebs-Ringer's solution, as compared to controls weighed and ashed immediately

TABLE III

SULPHATE CONTENT OF PUPPY CARTILAGE SLICES AS A FUNCTION OF TEMPERATURE

Sulphate Content of Cartilage Slices Immediately after Slicing in Krebs-Ringer's Solution at 37 Degrees Centigrade (*Milligrams of sulphate sulphur per 100 grams of cartilage*)	Sulphate Content of Cartilage Slices after Two Hours in Krebs-Ringer's Solution at 30 to 32 Degrees Centigrade (*Milligrams of sulphate sulphur per 100 grams of cartilage*)
364	183
370	132
429	149
336	132
404	121
419	
Average: 387 ± 39*	Average: 143 ± 24*

* Mean ± standard deviation.

TABLE IV

THE EFFECT OF IODOACETIC ACID AND SODIUM CYANIDE ON S^{35} UPTAKE AND TOTAL SULPHUR OF COSTAL-CARTILAGE SLICES FROM A FIVE-MONTH-OLD PUPPY *†

Inhibitor	Milligrams of Sulphate Sulphur Incorporated from S^{35}-Labeled Solution per 100 Grams of Cartilage	Standard Deviation	Total Sulphate (*Milligrams of sulphate sulphur per 100 grams of cartilage*)	Standard Deviation	t Values
None	0.664	0.053	351	37.4	
Iodoacetic acid (1×10^{-3} moles per liter)	0.404	0.035	371	18.0	1.53
Sodium cyanide (1×10^{-3} moles per liter)	0.456	0.026	405	26.0	3.85

* All values mentioned here are the average of ten samples. Sulphur determinations were done in quadruplicate; t values were calculated by comparing the iodoacetic acid and sodium cyanide sets of values with the controls without inhibition; t for sodium cyanide samples is significant, being higher than 3, thus indicating that total sulphur in sodium cyanide samples is higher than in controls.

† Twelve-hour incubation time.

TABLE V

$S^{35}O_4$ LOSS INTO INCUBATION SOLUTIONS FROM COSTAL CARTILAGE OF PUPPIES, TWO AND THREE MONTHS OLD *

Solution	Counts per Minute per Milligram of Cartilage		Percentage of Control	
	Experiment No. 1	Experiment No. 2	Experiment No. 1	Experiment No. 2
Control: Krebs-Ringer's solution	346	134	100	100
Krebs-Ringer's solution and 1×10^{-3} moles sodium cyanide	246	95	71	71
Krebs-Ringer's solution and 1×10^{-3} moles iodoacetic acid		131		98

* Twelve-hour incubation.

TABLE VI

THE EFFECT OF IODOACETIC ACID ON S^{35} UPTAKE BY COSTAL CARTILAGE FROM AN EIGHT-MONTH-OLD PUPPY AND ITS REVERSAL BY GLUTATHIONE *†

Inhibitor	Milligrams of Sulphate Sulphur Incorporated from S^{35}-Labeled Solution per 100 Grams of Cartilage	Standard Deviation	Total S (*Milligram per 100 grams of cartilage*)	Standard Deviation
None	1.07	0.06	430	26
Iodoacetic acid (1×10^{-3} moles per liter)	0.662	0.018	373	56
Iodoacetic acid and glutathione (4×10^{-3} moles per liter)	0.836	0.068	400	52
Glutathione (4×10^{-3} moles per liter)	1.02	0.09	387	51
Heated samples	0.811	0.086	415	33
Initial values			594	132

* Each value is the average of six determinations.
† Twelve-hour incubation.

after slicing. Sulphate loss increased drastically if the incubation temperature was decreased. In Table III is shown the sulphate content of cartilage slices maintained for two hours in Krebs-Ringer's solution at 30 to 32 degrees centigrade. Total sulphate content of cartilage slices immediately after slicing varied greatly from one experiment to another, and was due to age differences between the puppies used.

Uptake of S^{35} Sulphate

The uptake of S^{35} by cartilage slices in labeled Krebs-Ringer's solution was rapid during the first three hours, but after this period it was slower (Table I and Fig. 1). After thirty hours of incubation, the S^{35} content of the slices decreased, probably due to death of the tissue. Similar characteristics are shown by the control curve in Figure 2. Although some of this uptake can be from adsorption, other workers [2,4] have shown that most of it is incorporated into ester sulphate.

The Effect of NaCN on S^{35} Uptake and Total Sulphate

Sodium cyanide (1×10^{-3} moles per liter) inhibits S^{35} uptake by cartilage slices (Fig. 2). Total sulphate determinations performed on sodium-cyanide-poisoned tissue show an inhibition of the loss always observed in cartilage slices *in vitro*. Table IV refers to a twelve-hour experiment in which this protective effect against sulphate loss was observed. Heated samples show much the same results (Table VI).

Loss of Sulphate from Labeled Cartilage

The loss of $S^{35}O_4$ by labeled cartilage slices into non-radioactive Krebs-Ringer's solution was studied in order to verify the protective effect offered by sodium cyanide against total sulphate loss. The cartilage slices used were obtained from four-month-old puppies which had been injected intraperitoneally with eight millicuries of $Na_2S^{35}O_4$ and were sacrificed four days later. It was shown by Boström and Månsson that by this time practically all of the $S^{35}O_4$ in the cartilage is present as chondroitin sulphate. The results are shown in Table V, and confirm our previous findings. The values so obtained are actually the sum of all S^{35} lost from the slices, whether organic or inorganic.

It is of interest that the loss of sulphate sulphur determined by our usual chemical method and by estimation of the S^{35} in the cartilage before and after incubation was fourteen per cent by both methods.

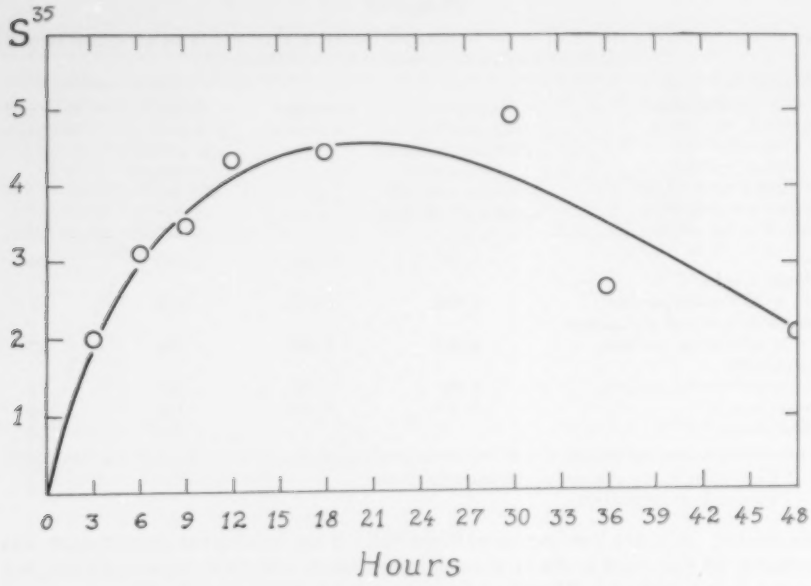


FIG. 1

The uptake of S^{35} by costal-cartilage slices from a two-month-old puppy.
Ordinate: S^{35} uptake × 10 expressed as milligrams of sulphate sulphur incorporated from S^{35}-labeled solution per 100 milligrams of cartilage. Each point is the average of two determinations.

The Effect of Iodoacetic Acid (IAA) and Its Reversal by Glutathione (GSH)

The inhibition of S^{35} uptake by cartilage poisoned with iodoacetic acid and other -SH inhibitors has been observed by Boström and Månsson who also showed that glutathione partially reverses this inhibition. Our findings (Table VI) confirm their results. Total sulphate loss does not seem to be markedly affected by iodoacetic acid or glutathione, or both (Tables IV, V, and VI).

DISCUSSION

Most investigators in the field [3,4,6,7] claim that incorporation of $Na_2S^{35}O_4$ into cartilage slices incubated in Krebs-Ringer's or similar solutions involves synthesis of chondroitin sulphate. Incorporation of S^{35} sulphate into chondroitin sulphate has been conclusively shown by Boyd and Neuman and Boström and Månsson. Although we have confirmed this uptake of S^{35}, the amount of it is small when compared with the loss of total sulphate. The true picture of the metabolic activity of the ground substance under these conditions is primarily one of degradation rather than synthesis. The extreme sensitivity of cartilage sulphate to decreases in temperature is evidence of the rapid alterations possible in the ground-substance gel when external conditions are not kept rigorously constant. Chondroitin sulphuric acid appears to be in a state of dynamic equilibrium. A possible mechanism of the state of negative balance which exists under these experimental conditions may be summarized as follows*:

$$\underset{\text{substrate}}{\text{Mucopolysaccharide}} + \text{"Active" } SO_4^{=} \xrightarrow[(1)]{} \text{CSA} \xrightarrow[(2)]{} \underset{\text{products}}{\text{Degradation}} + SO_4^{=}$$

Cellular enzyme activity has been shown by several investigators [2,4,6,7,15] to be necessary in step 1, the sulphation process. Many enzyme inhibitors, including

* In the formula, "Active" $SO_4^{=}$ is adenosinediphosphosulphate.

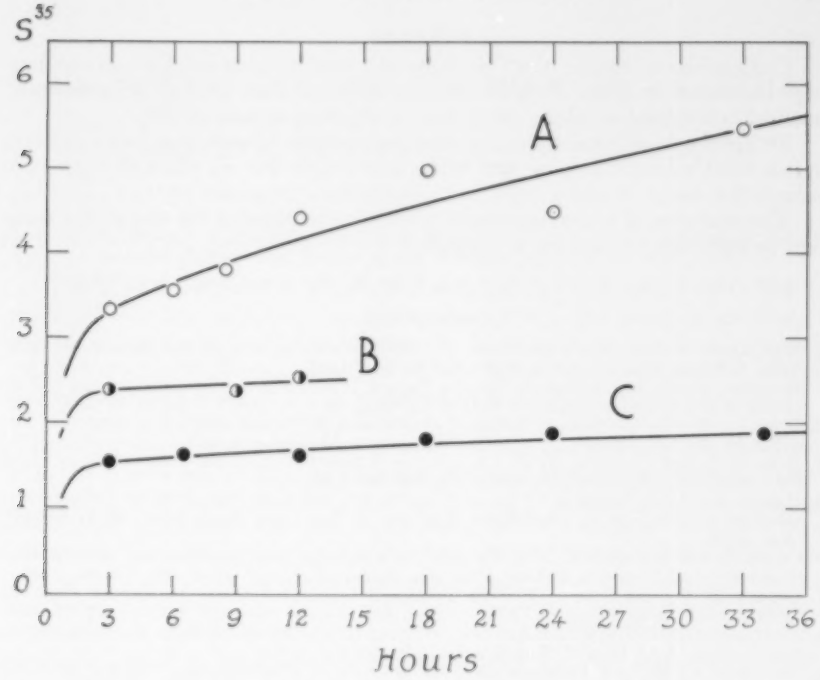


FIG. 2

The uptake of S^{35} by costal-cartilage slices from an eight-week-old puppy.
Ordinate: S^{35} uptake expressed as milligrams of sulphate sulphur incorporated from S^{35}-labeled solution per 100 milligrams of cartilage.
A: control samples incubated in Krebs-Ringer's solution.
B: samples heated for one hour at 45 degrees centigrade, then incubated as shown.
C: samples incubated in Krebs-Ringer's solution + 1×10^{-3} moles per liter sodium cyanide.
Each point is the average of two determinations.

cyanide and iodoacetate, have been found to decrease the uptake of S^{35}.

The enzymatic control of step 2 is suggested by the difference in effects of cyanide and iodoacetate. The latter decreases the uptake of S^{35} (step 1) without diminishing the loss of total sulphur (step 2). The addition of glutathione partly reverses the first effect. In cyanide poisoning and with heated samples, S^{35} uptake was also inhibited but total sulphate was protected against severe loss. The agent responsible for the desulphation of chondroitin sulphuric acid, possibly a sulphatase (step 2), seems to be cyanide-sensitive and thermolabile. Up to the present, despite many attempts [8], no direct evidence for chondrosulphatase activity has been demonstrated in cartilage. Soda, Katsura, and Yoda isolated a chondrosulphatase from shellfish liver, and similar enzyme activity has been noted in bacteria [16]. Both Dziewiatkowski [12] and Dohlman [8] presented indirect evidence that the rat is able to sever sulphate from injected chondroitin sulphate. Although our findings appear to suggest the possible presence of chondrosulphatase in cartilage, inhibition by sodium cyanide of another step in the breakdown of the chondroitin-sulphate complex could equally well explain our results. For example, splitting of the chondroitin sulphuric acid-protein linkage might well leave the sulphate more susceptible to loss.

Enzymatic breakdown of cartilage may be involved in the early stages of degenerative arthritis. A statistically significant elevation of the inorganic joint-fluid sulphate above the serum sulphate in cases of degenerative osteo-arthritis was presented in the first paper of this series [5]. The present work on total sulphur loss from incubated cartilage slices indicates a possible source of this sulphate.

SUMMARY

Comparison of uptake of S^{35} sulphate and total sulphur sulphate in cartilage strips incubated in Krebs-Ringer's solution indicates that over-all breakdown of chondroitin sulphate is taking place despite vigorous uptake of S^{35}.

S^{35} uptake is inhibited by cyanide and iodoacetate; however, cyanide protects against total sulphate sulphur loss while iodoacetate has no effect or may even increase the loss.

The existence of a cyanide-sensitive enzyme responsible for one of the early steps in cartilage breakdown is suggested.

NOTE: The careful technical work of Mrs. Mary Mueller is gratefully acknowledged.

REFERENCES

1. BLIX, GUNNAR, and SNELLMAN, OLLE: On Chondroitinsulfuric Acid and Hyaluronic Acid. Arkiv f. Kemi, Mineral., Geol., **A19**: No. 32, 1-19, 1945.
2. BOSTRÖM, HARRY, and MÅNSSON, BENGT: Factors Influencing the Exchange of the Sulfate Group of the Chondroitinsulfuric Acid of Cartilage in vitro. Arkiv f. Kemi, **6**: 23-37, 1953.
3. BOSTRÖM, H.: On the Sulfate Exchange of Sulfomucopoly-saccharides. *In* Connective Tissue in Health and Disease, pp. 97-102. Copenhagen, Munksgaard, 1954.
4. BOYD, E. S., and NEUMAN, W. F.: Chondroitin Sulfate Synthesis and Respiration in Chick Embryonic Cartilage. Arch. Biochem., **51**: 475-486, 1954.
5. CHRISMAN, O. D.; BECHTOL, C. O.; COELHO, R. R.; and BRENNAN, ROBERT: Sulphur Metabolism in Cartilage. I. Joint-Fluid Sulphate. J. Bone and Joint Surg., **40-A**: 457-461, Apr. 1958.
6. CLARK, I., and UMBREIT, W. W.: The Effect of Cortisone and other Steroids upon *in vitro* Synthesis of Chondroitin Sulfate. Proc. Soc. Exper. Biol. and Med., **86**: 558-561, 1954.
7. CURRAN, R. C., and GIBSON, T.: The Uptake of Labelled Sulphate by Human Cartilage Cells and Its Use as a Test for Viability. Proc. Roy. Soc., London, Ser. B, **144**: 572-576, 1956.
8. DOHLMAN, C. H.: The Fate of the Sulfate Group of Chondroitin Sulfate after Administration to Rats. Acta Physiol. Scandinavica, **37**: 220-234, 1956.
9. DOHLMAN, C. H., and FRIEDENWALD, J. S.: Pitfalls in Histochemistry: Unsuccessful Attempts to Demonstrate Mucoid Sulfatase Activity in Mammalian Tissue. J. Histochem. and Cytochem., **3**: 492-494, 1955.
10. DZIEWIATKOWSKI, D. D.: Isolation of Chondroitin Sulfate-S^{35} from Articular Cartilage of Rats. J. Biol. Chem., **189**: 187-190, 1951.
11. DZIEWIATKOWSKI, D. D.: Radioautographic Studies of Sulphate-Sulphur (S^{35}) Metabolism in the Articular Cartilage and Bone of Suckling Rats. J. Exper. Med., **95**: 489-496, 1952.
12. DZIEWIATKOWSKI, D. D.: Some Aspects of the Metabolism of Chondroitin Sulfate-S^{35} in the Rat. J. Biol. Chem., **223**: 239-249, 1956.
13. FISTER, H. J.: Manual of Standardized Procedures for Spectrophotometric Chemistry. New York, Standard Scientific Supply Corp., 1950.
14. LAYTON, L. L.: Labeled Inorganic Sulfate in the Diagnosis of Cartilaginous Tumors and Their Metastases. Cancer, **2**: 1089-1092, 1949.
15. LAYTON, L. L.: In vitro Sulfate Fixation by Granulation Tissue and Injured Muscle Tissue from Healing Wounds. Proc. Soc. Exper. Biol. and Med., **73**: 570-572, 1950.
16. NEUBERG, C., and HOFMANN, E.: Enzymatischer Abbau von Chondroitinschwefelsäure. Biochem. Zeitschr., **234**: 345-346, 1931.
17. SODA, T.; KATURA, T.; and YODA, O.: Glucosulfatase. XXII. Purification of Glucosulfatase and Chemical Analysis of the Purified Enzyme. Separation of Glucosulfatase from Chondrosulfatase. Enzymic Hydrolysis of the Sulphuric Acid Esters of Kojic Acid. J. Chem. Soc. Japan, **61**: 1227-1232, 1940.

Late Appearance and Evolution of a Cortical Defect in a Boy with Delayed Puberty

BY GERHART S. SCHWARZ, M.D., NEW YORK, N. Y.

From the Department of Radiology, College of Physicians and Surgeons, Columbia University, and the Radiological Service of the Presbyterian Hospital, New York

The term cortical defect is purely descriptive and was coined by Caffey for transitory cystlike rarefactions in the cortices of tubular bones of children. It replaces a nomenclature which includes the terms solitary xanthoma, cartilage rest, non-osteogenic fibroma, localized fibrous lesion, and subperiosteal desmoid tumor because the lesion is rarely subjected to and hardly ever merits biopsy.

The age incidence at which this lesion appears is highest between the fourth and twelfth years, with a sharp decline at the onset of puberty. The maximum incidence is reached at the age of eight years in both sexes. Cortical defects have never been observed during the first twenty-four months of life and only occasionally have been observed in adolescence[2,3,6]. Although there was some controversy about the maximum age at which the presence of cortical defects can be observed, there was, heretofore, no controversy about the fact that cortical defects never are first apparent after the fourteenth year of life. I wish to report, nevertheless, a case of delayed puberty in which a cortical defect was first apparent, as proved by roentgenograms, in a patient who was eighteen years old; roentgenograms made four months earlier showed no lesion. As is common in the detection of cortical defects, the roentgenograms demonstrating the defect were made for the study of another, unrelated lesion present in the same bone—an osteoid osteoma. The detection of the cortical defect was purely incidental.

E.S., a white boy, was first seen in the Vanderbilt Clinic of the Presbyterian Hospital on October 7, 1953, when he was seventeen years and eight months old, because of underdevelopment (his fourteen-year-old brother towered over him). In school the patient noticed that he had weighed less and had been smaller than his classmates since the age of thirteen; before that time he seemed to be on a par with his friends of the same age. His muscles seemed to him to be weak and poorly developed. (The boy's father did not shave until the age of twenty-five.)

The physical examination revealed a weight of seventy-two pounds and a height of sixty-three and one-half inches. His muscular development was below average. Pubic hair was sparse but of normal distribution. The testicles and penis were of normal size. Both testes were descended and of normal consistence. Facial and axillary hair were missing, and the change of voice had not occurred. The pertinent laboratory findings, including the erythrocyte sedimentation rate, iodine uptake, the Mazzini test for syphilis, the calcium and phosphorus levels, and the alkaline phosphatase, were within normal limits. The excretion of 17-ketosteroids in twenty-four hours was 4.02 milligrams.

A testicular biopsy was performed on January 5, 1954. It revealed the tubules to be normal in size and contour showing all stages of spermatogenesis. Spermatozoa were present, but rare. Interstitial cells were sparse but normal in appearance. The estimated developmental age of the testes was twelve to thirteen years—that is, five to six years below the patient's chronological age. A histological diagnosis of an otherwise normal testis was made. By February 23, 1954, the patient had gained nine pounds. Several spontaneous emissions had occurred. By May 18, 1954, the patient's height had risen to sixty-four and one-half inches, and by December 14, 1954, his weight had progressed to ninety-seven and one-half pounds. The endocrinologist examined the patient on that date and stated that he was definitely in puberty and referred him to the Orthopaedic Clinic for further follow-up.

ROENTGENOGRAPHIC FINDINGS

Roentgenograms of the right femur were made on admission because of a painful lesion in the upper part of the shaft which was diagnosed as osteoid osteoma and curetted. (This procedure was followed by permanent cessation of pain and a clinical cure. The diagnosis was confirmed histologically except for one dissenting opinion favoring low-grade osteomyelitis.)

FIG. 1

FIG. 2

Fig. 1: Admission roentgenogram of the lower end of the right femur when the patient was seventeen years and eight months old. No cortical defect is present.

Fig. 2: Lateral view of the right femur made on September 28, 1954, when the patient was eighteen years and seven and three-quarters months old. This roentgenogram was the first to show the cortical defect. The distal femoral epiphysis is still open, which is normal for this age.

FIG. 3-A

FIG. 3-B

Fig. 3-A: Anteroposterior view of the distal part of the right femur made on September 13, 1955, when the patient was nineteen years and nine months old. For all practical purposes, the epiphysis has closed. This constitutes a normal, even somewhat early, closure and is entirely consistent with the puberty now manifested. (See Fig. 3-B for comment on the cortical defect.)

Fig. 3-B: Lateral roentgenogram of the distal part of the right femur made on same date as Fig. 3-A. The cortical defect has become larger and now has a thin sclerotic margin. It lies in the mediposterior cortex of the shaft.

The pertinent point was that no cortical defect was demonstrable on the first set of roentgenograms in the region where a defect later appeared (Fig. 1). The skeletal age of the patient was estimated to be fourteen years at that time because the marginal epiphyses of the pelvis had not as yet ossified and none of the epiphyses of the hands or wrists had closed. This age estimation was in good agreement with the known ages shown in roentgenograms of the hands and knees in atlases on skeletal development[5,7].

The first roentgenogram which exhibited a cortical defect was made on September 28, 1954, when the patient was eighteen years and seven and three-quarters months old. The defect then consisted of a cavity the size of a pinhead without definitive borders (Fig. 2). When the patient was nineteen years and nine months old (September 13, 1955), roentgenograms disclosed a much larger cystlike rarefaction in the same region, which now possessed a sharply delineated, thin sclerotic margin (Figs. 3-A and 3-B). Finally, on December 3, 1958, when the patient was twenty-two years and ten months old, the last roentgenogram (Fig. 4) showed that the cortical defect had become bicameral and was surrounded by a wider sclerotic margin which was thickest at its medial and lateral aspects. Although a cephalad migration (away from the epiphysis) was described by Caffey, as well as by Ponseti and Friedman, as characteristic of some defects of the cortex of the femur, this did not occur here.

FIG. 4

Anteroposterior view of the right femur, made on December 3, 1958, when the patient was twenty-two years and ten months old. The cortical defect is now surrounded with a thicker sclerotic zone and is bicameral.

COMMENT

Although this patient's chronological age was eighteen years and seven and three-quarters months when the cortical defect appeared, his skeletal age by roentgenogram was only fourteen to fifteen years, and his histological testicular age was even less, being only twelve to thirteen years. His skeletal age at the time of onset of this defect coincides with the latest chronological age at the onset of this condition which has been reported in the literature. This may implicate an endocrine mechanism as a possible causative or contributing factor in the formation of these lesions. The affected leg in this patient remained approximately three-quarters of an inch longer than the opposite extremity during the period of observation. This discrepancy was attributed to stimulation of growth by the osteoid osteoma. To date, no reports have appeared correlating accelerated longitudinal growth with these cortical defects.

REFERENCES

1. Allen, D. H.: A Variation of Diaphyseal Development which Simulates the Roentgen Appearance of Primary Neoplasm of Bone. Am. J. Roentgenol., **69**: 940-943, 1953.
2. Caffey, John: On Fibrous Defects in Cortical Walls of Growing Tubular Bones: Their Radiologic Appearance, Structure, Prevalence, Natural Course, and Diagnostic Significance. Adv. Pediat., **7**: 13-51, 1955.
3. Greulich, W. W., and Pyle, S. I.: Radiographic Atlas of Skeletal Development of the Hand and Wrist. Ed. 2. Stanford, Stanford University Press, 1959.
4. Hatcher, C. H.: The Pathogenesis of Localized Fibrous Lesions in the Metaphyses of Long Bones. Ann. Surg., **122**: 1016-1030, 1945.
5. Jaffe, H. L., and Lichtenstein, Louis: Non-osteogenic Fibroma of Bone. Am. J. Pathol., **18**: 205-221, 1942.
6. Ponseti, I. V., and Friedman, Barry: Evolution of Metaphyseal Fibrous Defects. J. Bone and Joint Surg., **31-A**: 582-585, July 1949.
7. Pyle, S. I., and Hoerr, N. L.: Radiographic Atlas of Skeletal Development of the Knee: A Standard of Reference. Springfield, Illinois, Charles C. Thomas, 1955.
8. Sontag, L. W., and Pyle, S. I.: The Appearance and Nature of Cyst-Like Areas in the Distal Femoral Metaphyses of Children. Am. J. Roentgenol., **46**: 185-188, 1941.

DISCUSSION

Plantar Dissection

(Continued from page 164)

dissection. Finally, I would warn of the difficulty in identifying the joints of the foot in a young child. This operation should be performed slowly, deliberately, and carefully. The operation is really an exercise in the anatomy of the sole of the foot.

Clarence Heyman, M.D., Cleveland Heights, Ohio: I know that Dr. Bost is disturbed about the overcorrection in valgus position. He did not say what he did with the posterior tendon after he released it. I have found in medial releases that if I do tenotomy of the posterior tendon, I am quite sure that valgus of the ankle and overcorrection will result. So rather than sacrifice it, I meticulously lengthen it; I think in that way I have materially decreased the number of overcorrections or valgus of the ankle.

Dr. Bost (closing): Dr. Garceau's discussion certainly reveals that he knows a great deal about club-foot. He has hit it on the head when he says that we agree that much of the fault in correction lies with the surgeons. We either are not correcting these tight ligaments sufficiently or we are overcorrecting them. I have been in the habit of merely dissecting the tendon free and letting it lie and reattaching where it will.

I thank Dr. Heyman for his suggestion. I personally have felt that the reason for overcorrection in valgus position was overenthusiasm in the way that we released the medial structures, particularly the deltoid ligaments. We are trying to correct this in our subsequent series. We have now performed this operation over 200 times on another series of patients, many of them one or two years old. These patients are not now old enough for us to give a final assessment; I am not even sure that our results are any better than those we have reported. I certainly hope that they are.

Dr. Garceau hit another nail right on the head. I, too, think it is wrong to do the anterior tibial transplant at the same time that we do this extensive operation. I think it would be better to wait before doing the transplant and, of course, if a transplantation of the anterior tibial tendon alone will suffice, we would not do this operation. I hope that we know when not to do it.

Simple Night Splint for Correction of Tibial Torsion in Children

BY REX J. HOWARD, M.D., AND L. P. KLEUSER, M.D., FORT WORTH, TEXAS

In the treatment of tibial torsion in children Kite [1] emphasized the importance of preventing positions during sleep which would tend to accentuate the deformity. The purpose of this communication is to present a simple device to prevent these attitudes or positions. This device was suggested by the parents of one of our

FIG. 1

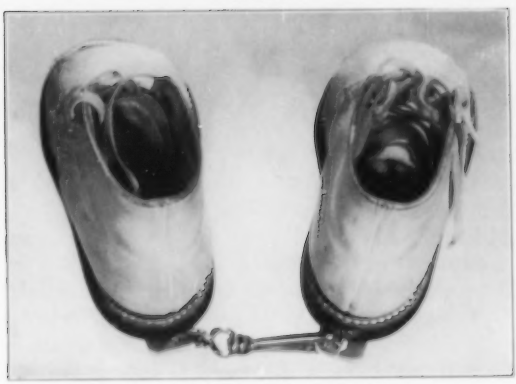


FIG. 2

patients and further developed by us as a simple and effective method for the treatment of tibial torsion by a night splint.

This splint came into being after a discussion of the problem of external tibial torsion with a patient's mother. She and her husband developed the apparatus by punching a hole in the toe of each shoe with an ice pick and fastening the shoes together with a key chain.

This splint was modified and further adapted for tibial torsion by securing

the shoes together either at the toes or at the heels using metal bars attached to the shoes and a snap to attach these bars together (Figs. 1 and 2). This device is not new, but is a modification of existing methods which we have found to be advantageous.

One advantage is that the fixation is not so rigid as that with a Denis Browne splint. As a result, it seems to bother the mother less, since the shoes can be fastened together after the child is asleep. Although this form of treatment of tibial torsion can be carried out very well by the Denis Browne splint, we have found this simple splint to be more convenient and just as effective. Two important considerations are: (1) it can be used in the winter when blankets are needed since it does not become tangled in the bed coverings as does a Denis Browne splint, and (2) it is less expensive to prepare and can be made by either a brace maker or the parents using an old pair of shoes with the toes cut out.

We have been using this splint for three years with satisfactory results. By placing the metal bars on the front of the sole or the back of the heel we have used the splint for either external or internal tibial torsion. It has proved equally effective in both conditions. In general, when the parents have been cooperative adequate correction has been achieved without the need for shoe corrections after the splint has been worn at night for three or four months.

1. Kite, J. H.: Personal communication.

Coxa Plana in Monozygotic Male Twins

BY COMMANDER ADOLPHUS W. DUNN, *Medical Corps, United States Navy*

From the United States Naval Hospital, Guam, Mariana Islands

There are few reports in the literature of coxa plana occurring in twins. In 1954 Giannestras reported the occurrence of Legg-Perthes disease in identical male twins, and the concomitant presence of coxa plana in a younger sister. In a review of the literature at that time he found no reports of true Legg-Perthes disease occurring in twins without other stigmata of diffuse epiphysitis. Derian, in 1956, presented a study of coxa plana in dizygotic male twins. In 1957 Söderberg reported the simultaneous occurrence of coxa plana in enzygotic twins and suggested that there might be reason to suppose a congenital causal factor in these cases. In 1959 Wansbrough, Carrie, Walker, and Ruckerbauer presented a study of the genetic aspects of coxa plana and mentioned the occurrence of bilateral Legg-Perthes disease in each of monozygotic male twins. They stated that the pattern of inheritance based on the present pedigrees was found by Ruckerbauer to be a sex-influenced, autosomal dominant with reduced penetrance and varying expression. Statistical analysis did not support a recessive mode of inheritance.

Familial occurrence of coxa plana was reported by Hagen in 1939, Lindemann in 1941, McComas in 1946, Stephens and Kerby in the same year, Hamsa and Campbell in 1953, and Goff in 1954. In Lindemann's report the femoral-head changes were associated with bilateral coxa vara.

FIG. 1-A

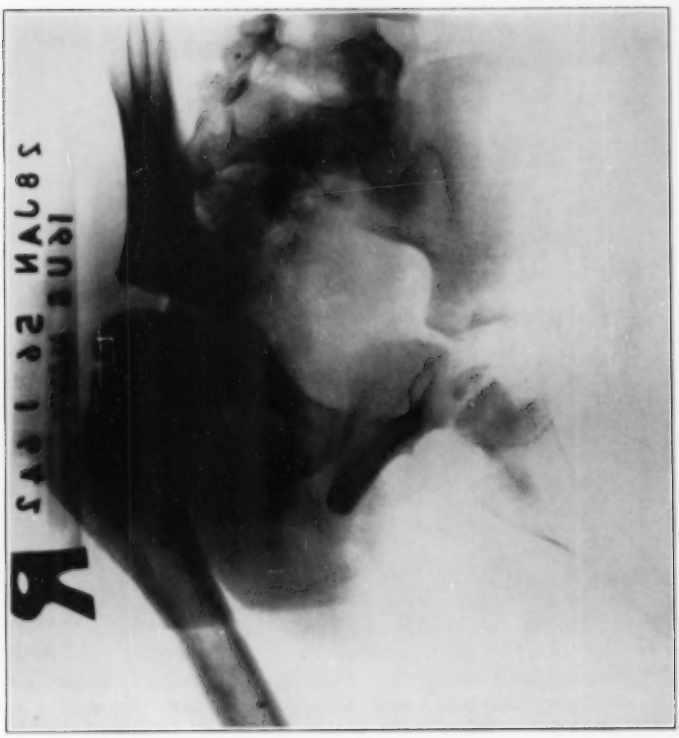


FIG. 1-B

Figs. 1-A and 1-B: Twin Donald R. Roentgenograms made on January 28, 1956, showing coxa plana in degenerative stage on the left.

This case report is of the simultaneous occurrence of coxa plana in monozygotic male twins. Although fingerprint studies and cross skin-grafting were not done, the facts that these boys were similar in height, weight, facial appearance, texture and color of skin and hair, color and pattern of iris, and had identical

FIG. 2-A

FIG. 2-B

Figs. 2-A and 2-B: Twin David R. Roentgenograms made on February 17, 1956, showing Legg-Perthes disease on the left.

major and minor blood groups indicate that they are enzygotic twins. The results of blood typing in both twins were as follows: major group-÷-A. Rh groups: CDe—c?e; M factor=positive; and N factor=negative. No anti-d serum was available. Hence, the question mark in the Rh typing. In one of the twins, Donald R., the left hand is dominant and in the other, David R., the right hand is dominant.

There is no familial history of any hip disease, and roentgenographic examination of the hips of two male siblings—one three years old and the other ten years old—showed no abnormality. Both twins had been in good general health and neither had had any significant illness. They had not had any trauma.

Donald R., five and one-half years old, was seen on January 27, 1956, because his parents had

FIG. 3-A

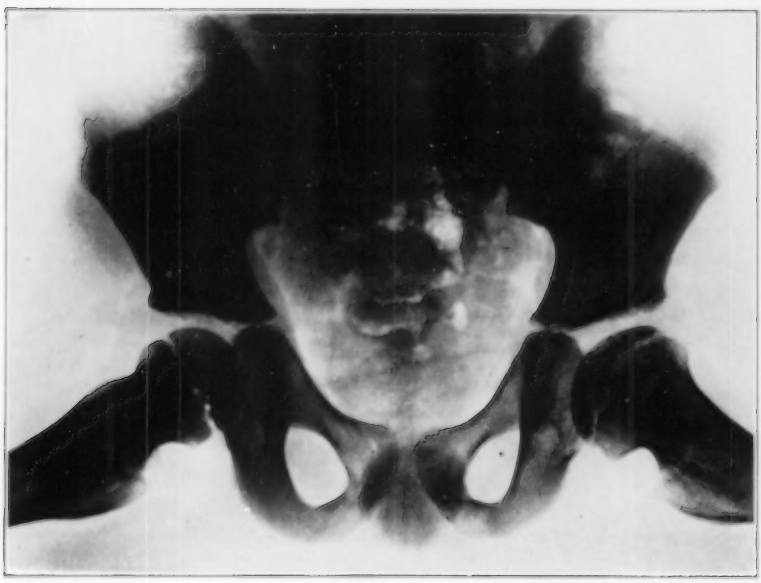


FIG. 3-B

Figs. 3-A and 3-B: Donald R. Roentgenograms made on August 18, 1958, showing healed coxa plana on the left.

noted that he limped on the left and they thought that there was shortening of his left lower extremity. One year previously he had been seen by a pediatrician because of a tendency to pull in his left leg when he walked. Roentgenographic examination of the feet was made, and medial heel wedges for his shoes were prescribed. He was not seen by an orthopaedic surgeon at that time. During the intervening year he had had a slight limp on the left when running, but this was not noticed when he walked. At no time did he complain of pain in the hip, thigh, or knee. Examination on January 27, 1956, revealed 30 per cent limitation of

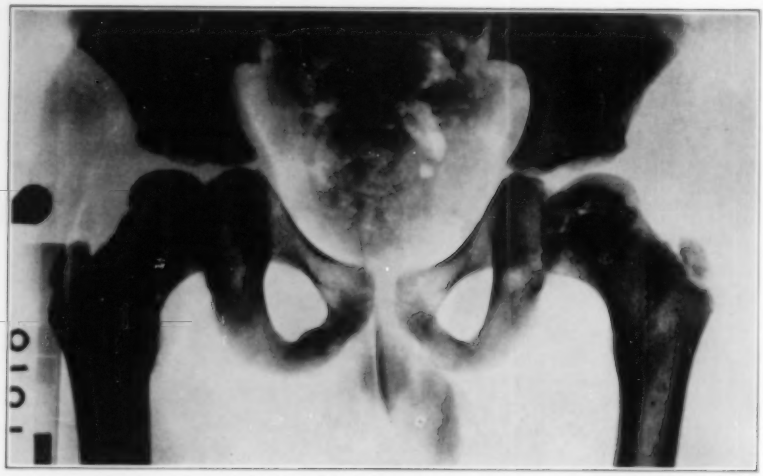


FIG. 4-A

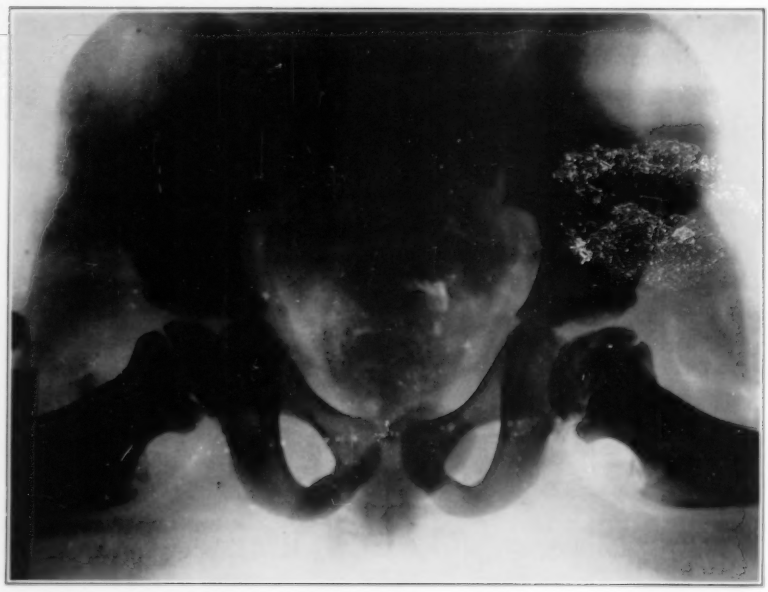


FIG. 4-B

Figs. 4-A and 4-B: David R. Roentgenograms made on May 29, 1958, showing regeneration of the left capital femoral epiphysis.

abduction and slight restriction of internal rotation of the left hip. There was full range of motion of the hip in external rotation, flexion, extension, and adduction. Discomfort was experienced on abduction and internal rotation. He walked with a barely perceptible limp on the left. Roentgenograms revealed coxa plana on the left (Figs. 1-A and 1-B). Laboratory findings were within normal limits. Treatment was with a patten-bottom brace until July 1956 when a change was made to an ischial weight-bearing brace with a functional knee hinge. Serial roentgenograms showed the usual progression to healing of coxa plana (Figs. 3-A and 3-B). Full weight-bearing was permitted thirty months after treatment was instituted. At that time his gait was normal, and motion in the left hip was neither restricted nor painful.

The parents requested examination of the hips of David R., Donald's identical twin, even though he had never had hip symptoms. David was seen in consultation on February 17, 1956.

Questioning of the parents elicited no history of any disturbance of gait, and, to their knowledge, David had at no time complained of pain in either of his lower extremities. No limp was detected on examination. There was full range of painless motion of both hips, and no muscle spasm was found. Roentgenograms showed changes indicative of coxa plana on the left (Figs. 2-A and 2-B). All laboratory examinations were within normal limits. Treatment consisted of a patten-bottom brace until July 6, 1956, when an ischial weight-bearing brace with functional knee hinge was substituted. When bracing was discontinued twenty-seven months after institution of treatment, his gait and mobility of the left hip were normal. Roentgenograms at that time showed complete regeneration of the left capital femoral epiphysis (Figs. 4-A and 4-B).

REFERENCES

1. Derian, P. S.: Coxa Plana in Dizygotic Male Twins. J. Bone and Joint Surg., **38-A:** 901-907, July 1956.
2. Giannestras, Nicholas: Legg-Perthes Disease in Twins. J. Bone and Joint Surg., **36-A:** 149-152, Jan. 1954.
3. Goff, C. W.: Legg-Calvé-Perthes Syndrome and Related Osteochondroses of Youth, p. 31. Springfield, Illinois, Charles C. Thomas, 1954.
4. Hagen, W. H.: Coxa Plana. Report of Two Bilateral Cases in Brothers. J. Bone and Joint Surg., **21:** 1028-1030, Oct. 1939.
5. Hamsa, W. R., and Campbell, L. S.: Osteochondritis Deformans Coxae Juvenilis. Familial Demonstration. Am. J. Dis. Child., **86:** 54-59, 1953.
6. Lindemann, Karl: Das erbliche Vorkommen der angeborenen Coxa vara. Zeitschr. f. Orthop., **72:** 326-352, 1941.
7. McComas, Elizabeth: Perthes's Disease and Its Occurrence as a Familial Condition. Med. J. Australia, **2:** 584-585, 1946.
8. Söderberg, Lennart: Simultaneous Occurrence of Coxa Plana in Enzygotic Twins. Acta Orthop. Scandinavica, **27:** 135-140, 1957.
9. Stephens, F. E., and Kerby, J. P.: Hereditary Legg-Calvé-Perthes' Disease. J. Hered., **37:** 153-160, 1946.
10. Wansbrough, R. M.; Carrie, A. W.; Walker, N. F., and Ruckerbauer, G.: Coxa Plana, Its Genetic Aspects and Results of Treatment with the Long Taylor Walking Caliper. A Long-Term Follow-up Study. J. Bone and Joint Surg., **41-A:** 135-146, Jan. 1959.

Proceedings

ASSOCIATION OF BONE AND JOINT SURGEONS

As Prepared by William H. Blodgett, M.D., Detroit, Michigan, *Chairman*,
Inter-Association Relationship Committee

The Eleventh Annual Meeting of the Association of Bone and Joint Surgeons was held in Washington, D. C., April 12 through April 15, 1959, under the Presidency of Dana Street, M.D., North Little Rock, Arkansas.

Officers elected for the coming year are:

President: Jack K. Wickstrom, M.D., New Orleans, Louisiana;
Vice-President: Frederick R. Thompson, M.D., New York, N. Y.;
Second Vice-President: Harry R. Walker, M.D., Oakland, California;
Treasurer: C. Fred Ferciot, M.D., Lincoln, Nebraska;
Secretary: William M. Deyerle, Richmond, Virginia.

The 1960 meeting will be held in Little Rock, Arkansas, with Dr. Dana Street acting as host.

A résumé of each paper presented follows:

Biomechanics of Hip Prosthesis. Dr. Duncan McKeever said that according to engineering principles the ideal prosthesis has a straight stem, with the head of the prosthesis resting on the cut surface of the neck at an angle between 15 and 20 degrees to the horizontal, and the distal end of the stem protruding through the cortex of the femur so that the stem bears no weight. Any curved-stem prosthesis is subject to torque during walking, which results in progressive loosening and settling of the prosthesis.

Dr. McKeever found results over a period of nine years with the prosthesis described to be excellent.

The paper was discussed by Dr. Fred Thompson, Dr. Fred Ferciot, Dr. Richard Reiley, and Dr. Dana Street.

Dysvascular Necrosis of the Head of the Femur. Dr. M. Beckett Howorth [1] discussed vascular disturbances of the femoral head. He said that the term *dysvascular necrosis* was more inclusive and more descriptive than either avascular or aseptic necrosis. Dysvascular necrosis of the capital epiphysis occurs in congenital dislocation of the hip, cretinoid epiphyseal dysgenesis, dyschondroplasia, coxa plana, slipping capital femoral epiphysis, osteochondritis dissecans, fracture of the femoral neck, fracture of the acetabulum, traumatic dislocation of the hip, caisson disease, Gaucher's disease, gout, and Charcot's disease. It also occurs as a sequel of roentgen, radium, or other radiation and in spontaneous osteolysis and may also be of unknown etiology.

Illustrative roentgenograms were shown of various phases of dysvascular necrosis.

Teaching of Orthopaedic Surgery as a Part of the Surgical Department at Georgetown University. Dr. Robert Coffey [2] described a new plan of teaching third and fourth year medical students that eliminates the didactic lecture method and substitutes a correlated program of panel presentations of individual subjects. Each subject is presented as a unit by a group representing the various relevant fields of medicine. As a result, a complete presentation of each subject is given at a single time.

Them Bones Will Rise Again. Dr. William Tobin [3] presented a series of lantern slides showing photographs of prehistoric bones with various pathological entities involving the spine and extremities. Photographs of series of skulls showed that surgical craniotomies had been done at some time before historical reports.

Bone Tumors. Dr. Murray Copeland [4] discussed parosteal osteomata or parosteal sarcomata using a series of cases for illustration.

Dr. Copeland said that these tumors may be benign or malignant and involve the lower end of the femur or either end of the humerus, as well as other sites in the long bone. There is broadening of the metaphysis and thickening of the cortex. Calcification independent of the bone may be seen in the region of the tumor.

1. Atlantic Street, Stamford, Connecticut.
2. 1150 Connecticut Avenue, N.W., Washington 6, D. C.
3. 1835 Eye Street, N.W., Washington 6, D. C.
4. 3900 Reservoir Road, N.W., Washington 7, D. C.

Most of the patients had had symptoms of aching pain over a period of months prior to the recognition of the condition. Treatment in these cases had consisted in local excision, x-ray therapy, and amputation, but Dr. Copeland felt that if there was any doubt as to whether the lesion was benign or malignant amputation is preferable to running the risk of allowing the development of metastasis.

Problems on Basic and Applied Research of the Bone Graft. Captain George Hyatt[5] discussed problems in connection with bone grafts and presented a movie showing an experiment in which the hibernating gland of a fetal mouse was transplanted to the subcutaneous area of a recipient mouse. The reaction of the cells in this gland, as observed through a mica window inserted in the skin, was employed to determine the effect of various elements of bone transplanted into the tissues adjacent to the gland.

Description of Animal Research in Relation to Heterografts. Dr. Surenda P. Gupta[6] described bone-grafting experiments which demonstrated that when heterografts were placed in a defect in the cortex of the long bone of a dog's extremity, they were revascularized and stimulated paravascular-bone formation more slowly than autografts in similar circumstances.

The Physiology of the Intervertebral Disc. Dr. Henry L. Feffer[7] discussed the pathology of the intervertebral disc, the mechanism of intervertebral-disc herniation, and the changes in the disc which accompany advancing age, including the progressive desiccation associated with the loss of mucopolysaccharide from the collagen complex.

Dr. Feffer felt that with discography it is possible to distinguish between degenerative changes and herniations of the disc. The value and limitations of intradiscal injections of hydrocortisone were also discussed.

Discography, Its Usefulness and Limitations. Dr. William K. Massie[8] illustrated, by slides, the degenerative process that takes place in the nucleus pulposus and annulus fibrosus with advancing age and presented a series of discograms of the lower three lumbar levels. These slides illustrated both normal and abnormal discograms, correlating the abnormal appearances with the pathological changes. Emphasis was placed on the importance of correlating the clinical symptoms, the location and degree of pain on injecting the contrast medium into the disc, and the appearance of the discogram. Myelography outlines a prolapsed disc more clearly and is more useful to the physician who limits his surgical treatment to this condition. It gives no information concerning the discs that have not protruded. Segmental fusion, which does not include all the interspaces that are involved, is relatively ineffective. In the patient whose age, sex, and occupation justify spine fusion, preoperative or operative discograms permit complete evaluation of the existing degenerative changes and form the basis for a rational decision as to the extent of the spine fusion. The injection of hydrocortone intradiscally is a fringe benefit of discography which provides temporary relief of symptoms in 75 per cent and remissions lasting over one year in 25 per cent of patients.

Osteoporosis in Bone Physiology. Dr. John J. Canary[9] discussed osteoporosis of bone as demonstrated by roentgenograms, pointing out that the calcium deficit of bone must be from 20 to 60 per cent before roentgenographic manifestation of the demineralization is evident.

The causes of osteoporosis were divided into seven categories: hypogonadism, Cushing's syndrome, disuse atrophy, malnutrition, old age, idiopathic conditions, and acromegaly. In osteoporosis the calcium infusion test is normal as are the levels of serum calcium and phosphate, and the citric-acid output. These findings are also little changed by hormone therapy although insulin has been shown to have an effect on calcium retention after calcium infusion.

Acute Congestive Failure in the Orthopaedic Patient. Dr. Jack Segal[10] pointed out that by the time a patient with early congestive heart failure has basal rales or edema, considerable decompensation is already present and that treatment with digitalis and a diuretic may be indicated as a preoperative measure. In orthopaedic practice an unusual cause of congestive heart failure is Paget's disease and strain of the left side of the heart caused by the multiple arteriovenous fistulae present in this disease.

Major Vascular Injuries Associated with Fractures. Dr. Charles Hufnagel[11] said that arterial injuries occur most frequently in association with fractures around the knee and

5. Naval Medical School, Bethesda 14, Maryland.
6. Calcutta, India.
7. 1703 Rhode Island Avenue, N.W., Washington 6, D. C.
8. 288 South Limestone Street, Lexington 4, Kentucky.
9. Georgetown University Hospital, Washington 7, D. C.
10. Georgetown University School of Medicine, Washington, D. C.
11. Veterans Administration Center, Los Angeles 25, California.

upper part of the humerus. The injuries include complete or partial division of the artery and traumatic thrombosis.

Complete division of the major vessels is apparent from the circulatory changes distal to them, whereas traumatic thrombosis is characterized by gradual loss of sensation and motor function of the distal portion of the extremity. Reconstruction of the vessel is indicated when continuity of blood flow has been lost. Vessel replacement or removal of the thrombosis should be carried out in the presence of progressive arterial thrombosis.

When there is trauma to the smaller vessels the associated sympathetic overflow is treated by sympathetic block or by sympathetic-nerve depressants. In the presence of arterial damage anticoagulants are preferred whenever circumstances permit their use.

Certain Aspects of Physiology of Bone. Dr. Fred C. Bartter [12] reviewed the pathological changes associated with metabolic bone disease, as well as the changes in the serum calcium and phosphorus and in the urinary excretion of calcium. In addition, a comparison was made of the changes occurring in osteomalacia and osteitis fibrosa cystica.

Experimental Transplant of Femoral Osteochondral Caps. Dr. Anthony DePalma [13] presented the results of experimental work in dogs in which cartilaginous caps with a small amount of underlying bone were fashioned from femoral heads and transplanted to the denuded femoral neck as osteocartilaginous grafts. Three types of graft were used: autogenous, fresh homogenous, and homogenous grafts that had been stored in plasma.

It was found that all the grafts were fixed in from six to twelve days and that in all of the autogenous grafts the cartilage and the bone structure were normal at the end of one year.

In some of the homogenous grafts of both types destruction of the bone occurred after thirty-six days, the bone being replaced by fibrous tissue. In some of these, the articular cartilage of the head was also destroyed. At the end of one year, in the fresh homogenous grafts, 15 per cent of the bone and 30 per cent of the cartilage had been destroyed. In the plasma-stored homogenous grafts, 4.3 per cent of the bone and 43.0 per cent of the cartilage were destroyed.

Histologic Evidence of Control of Growth in the Femur and Tibia by Staples. Dr. Charles W. Goff [14] described the findings in the selected biopsies of the lower femoral and upper tibial epiphyseal plates from over 200 children in whom these epiphyseal plates had been stapled for one reason or another. A small wedge-shaped section of bone and the epiphyseal plate were removed along with the staple, and microscopic studies were made of the specimen.

At the end of one year after stapling, not much change in microscopic architecture of the epiphyseal disc was apparent. At eighteen months there was distortion of the columnar alignment of the cartilaginous cells, and at two years a layer of bone formed adjacent to and parallel to the epiphyseal disc. Between thirty-four and thirty-six months after stapling the epiphyseal plate began to disintegrate and in some instances bone union developed between the epiphysis and the metaphysis. In these cases the arrest is permanent.

In three patients further growth in length occurred at the epiphyseal disc when the staples were removed after forty-four months. In the other patients in whom epiphyses were stapled for forty-four months total arrest of growth was observed after the staples were removed. About one-third of the epiphyseal plates stapled for less than forty-four months also showed total arrest after removal of the staples. Growth was measured by orthoroentgenogram and, in some instances, by metal markers inserted in the bones.

Previously Undescribed Clinical Bursitis about the Knee. Dr. Fred Stuttle [15] presented an anatomical description of the bursa underlying the medial collateral ligament of the knee and described it as the "no name, no fame bursa". In flexion the anterior edge of the ligament slides posterior to the bursa. Inflammatory changes in this bursa may produce a clinical picture similar to that associated with lesions of the medial meniscus. Such a bursitis may be initiated by mild trauma of the knee associated with a twisting injury. Full extension and external rotation of the tibia on the femur may be limited by pain, thus simulating the locking of the knee seen with a displaced medial meniscus. The bursa is located exactly at the joint line rather than below the joint line, as is sometimes demonstrated in anatomical textbooks. The most frequently effective treatment is injection of novocain and hydrocortisone into the bursa.

A Functional and Cosmetic Correction of Metatarsus Latus. Dr. Joseph Brown [16] said that excision of the fifth ray of the foot (the toe and the entire metatarsal) has proved to be

12. Clinical Center, National Institutes of Health, Bethesda, Maryland.
13. 248 South 21st Street, Philadelphia 3, Pennsylvania.
14. 30 Farmington Avenue, Hartford 5, Connecticut.
15. 1200 Hamilton Boulevard, Peoria 2, Illinois.
16. 10515 Carnegie Avenue, Cleveland 6, Ohio.

effective in the treatment of metatarsus latus or splay foot. The resultant narrowed foot eliminates the abnormal pressure phenomena usually associated with a splay foot in a woman.

The morbidity of the procedure has been slight, weight-bearing being allowed as soon as the patient desired. The function of the foot after the operation in these patients has been normal with no recurrences of corns, bunionettes, interdigital neuroma, or metatarsalgia, and no aggravation or accentuation of the hallux valgus deformity which, in many of the patients, was present before operation. The first patient in this series was operated on in 1948.

Giant-Cell Tumors of Tendon Sheaths in the Hand. Dr. George S. Phalan[17] reviewed fifty-seven cases from the files of the Cleveland Clinic of giant-cell tumors of tendon sheaths of the hand. The age incidence varied but the peak incidence was between the fourth and fifth decades of life. The tumors were more common on flexor surfaces. Bone involvement was not frequent and when it occurred it took the form of erosion secondary to compression.

The gross and microscopic characteristics of the tumors were discussed and illustrated. These tumors are benign, although local recurrence may occur, usually by the fourth to sixth month after operation, if complete excision of the tumor has not been accomplished. With the exception of ganglia, giant-cell tumors of tendon sheaths are the most common tumors in the hand.

Ununited Fractures of the Tibia. Dr. H. Relton McCarroll[18], the *Annual Guest Speaker*, reviewed the various methods for the surgical treatment of non-union of the tibia and presented a series of patients treated by a modified Phemister graft. Through an anterolateral approach both the tibia and the fibula were exposed. An attempt was made, whenever possible, to produce a synostosis between the fibula and the tibia by wedging cortical graft, obtained from the longer tibial fragment, between the fibula and the tibia. Cancellous onlay grafts were placed around the pseudarthrosis of the tibia without attempting to curette or to remove the fibrous tissue between the bone ends of the tibia.

Dr. McCarroll emphasized the importance of draining the wounds with a Penrose drain for from two to ten days to avoid the formation of a deep-seated hematoma. He also advised the use of a postoperative pressure dressing beneath the plaster cast, replacing this after ten days with a standard toe-to-groin cast.

In cases where there was an overlying ulcer with surrounding scar tissue the importance of preliminary excision of the scar and replacement with a full-thickness pedicle skin graft was emphasized. In a few instances this procedure alone caused sufficient increase in the blood supply to the fracture area for spontaneous union of the fracture to occur.

Low-Back Surgery and the Orthopaedist. Dr. Harry Walker[19].

Elective Orthopaedic Surgery in Two Severe Hemophiliac Patients. Dr. Thomas F. Broderick, Jr.[20], described his experience with two patients with severe hemophilia and consequent abnormalities of the joints. An extensive reconstructive surgical procedure was carried out successfully on each patient while the clotting time was controlled by whole-blood transfusions, antihemophiliac globulin, and beef-brain cephalin administered under the direction of a hematologist.

The paper was discussed by Dr. Anthony DePalma[21], who pointed out that surgery in hemophiliac patients is very dangerous unless it is done according to Dr. Broderick's method, in association with an excellent hematologist and with a laboratory equipped and staffed for the management of this type of patient.

Fractures of the Head and Neck of the Radius in Children under Twelve. Dr. Earl D. McBride[21] and Dr. Charles Monnet presented a review of twenty-nine fractures of the head and neck of the radius in children under twelve years of age treated at the McBride Clinic during the past fifteen years. Twelve of these patients have now reached maturity.

Severe displacement of the head of the radius can be accepted with expectation of an excellent result by the time growth is completed. Open reduction should be carried out early, and minimum reconstructive measures attempted. Delayed open reduction gave the poorest results. When significant displacement of the radial head persists, excision of the radial head after the completion of growth is the procedure of choice.

17. 2020 East 93rd Street, Cleveland 6, Ohio.
18. 3720 Washington Boulevard, St. Louis 8, Missouri.
19. 485 34th Street, Oakland 9, California.
20. 1265 Beacon Street, Brookline 46, Massachusetts.
21. 605 N.W. Tenth Street, Oklahoma City 3, Oklahoma.

Reconstruction of Rheumatoid Fingers. Dr. Frederick R. Thompson [22] demonstrated, with motion pictures, the operative technique for the correction of rheumatoid deformities of the fingers and hand, which was described originally by Riordan and Fowler. The ease with which the operation was performed and the great improvement in function attained by the patient in the early postoperative period were demonstrated. The difficulty in obtaining strength in the index finger comparable to that in the middle finger was emphasized. Suggestions were made as to how this difficulty could be overcome. The value of fusion of the interphalangeal thumb joint for added strength and the possibility of fusing the metacarpophalangeal joints of the second and third fingers were also discussed.

22. 16 East 90th Street, New York 28, N. Y.

NEW JERSEY ORTHOPAEDIC SOCIETY

The Annual Spring Meeting of the New Jersey Orthopaedic Society was held at the Hackensack Hospital, Hackensack, New Jersey, on Saturday, April 11, 1959, with Dr. Robert J. Neville as *Program Chairman* and *Moderator*. The program consisted of a combined Traumatic and Orthopaedic Clinical Day at the Hackensack Hospital in conjunction with the Spring Meeting of the New Jersey Orthopaedic Society. The guest of honor was Dr. William B. Estes, of Bethlehem, Pennsylvania, *President*, American College of Surgeons.

The Morning Session was arranged by the Department of Orthopaedic and Traumatic Surgery of the Hackensack Hospital and was devoted to the surgery of trauma in the emergency room. Dr. Spencer T. Snedecor, *Director*, presided.

The Management of a Complicated Traumatic Injury. Dr. Snedecor stressed the importance of teamwork, of efficiency, and personnel accustomed to handling problems of this type. He spoke of the need for a team captain to ensure proper organization by analyzing a situation immediately, calling the doctors needed to treat the patient. Members of the audience discussed the treatment of a patient with multiple injuries and the manner in which the various medical consultants were utilized with particular attention to liaison.

Non-Penetrating Abdominal Trauma was discussed by the guest of honor, Dr. William B. Estes.

The Morning Session was completed by an inspection, conducted by Dr. W. C. Rucker, of the Emergency Room facilities at the Hackensack Hospital.

The Afternoon Session was arranged by the New Jersey Orthopaedic Society and conducted by Dr. Robert J. Neville, *Program Chairman*. The following scientific papers were presented:

Etiology of Epiphyseal Displacement with Special Emphasis on the Capital Femoral Epiphysis. Dr. Peter J. Dulligan.

Flexion-Extension Injury of the Cervical Spine. Dr. Daniel B. Eck.

Follow-up Study. Fractures of the Neck of the Femur Treated by the McElvanney Procedure. Dr. Marshall Bergen.

Chondrosarcoma. Dr. Raphael R. Goldenberg and Dr. Philip Cohen.

The Scientific Session was followed by a Business Meeting of the New Jersey Orthopaedic Society. Dr. Keith E. Haines, *President*, presided.

DUNCAN CLARK McKEEVER
1905-1959

Duncan Clark McKeever was killed on the night of October 13, 1959, when a car plowed into him as he stood in the driving rain pouring gasoline into his automobile.

It is hard to realize that his talents have been taken away in this untimely fashion, before the many other fertile ideas within his mind were given to the world to help mankind. Duncan, to those who were close to him, was one of the brightest and best thinkers in contemporary orthopaedic surgery. His stimulating thoughts were constant challenges to all of us. We have lost a good friend. Orthopaedic surgery has lost one of its most talented originators.

He was born on September 13, 1905, in Valley Falls, Kansas. He attended Westport High School and Junior College in Kansas City, Missouri. Except for $50.00 given to him by his parents during his first year of medical school, he financed his education at the University of Kansas Medical School by his own efforts and graduated in 1929. Since he belonged to the Naval Reserve, the pattern of his internship was established. He spent one year at the United States Naval Hospital in Brooklyn, New York, and three years at the Great Lakes Naval Training Station near Chicago, Illinois. After completing his naval service he accepted a residency in pathology at St. Luke's Hospital in Kansas City, Missouri, where he came under the influence of Dr. Frank Dickson and Dr. Rex Diveley. These men soon converted Duncan from postmortems to Lane technique. After three years' preceptorship he became associated with them and continued to work with them until 1939 when he went to Houston, Texas, to establish his own practice.

Since he was in the Naval Reserve he was called to active duty in 1941. During the war his principal assignments were as Chief of Orthopaedics of the Long Beach Naval Hospital and later at the Area Hospital at Honolulu. He was discharged from the Service in 1945 with the rank of captain.

Duncan was certified in orthopaedic surgery in 1937, was a Fellow of The American Orthopaedic Surgeons; Fellow of the Clinical Orthopaedic Society; Fellow of the American College of Surgeons; Past-President of the Association of Bone and Joint Surgeons; Fellow of the American Medical Association; member of the Association of American Physicians and Surgeons; member of the Houston Orthopaedic Club and the Texas Orthopaedic Association; Co-Founder of the Spectators Correspondence Club; Civilian Orthopaedic Consultant for the Air Force; member of the American Society of Clinical Hypnosis; and currently President of the American Fracture Association.

Duncan was a member of the orthopaedic staffs of Memorial Hospital, St. Joseph's Hospital, Herman Hospital, and St. Luke's Hospital—all of Houston, Texas. He was past chief of the Orthopaedic Section of Memorial Hospital, past president of the Medical Staff of Memorial Hospital, Chief of Staff of the Texas Elks Crippled Childrens' Hospital, Ottine, Texas, a consultant for Queries and Answers Section of *The Journal of The American Medical Association;* and a director of The American Motor League (Safety).

Although Duncan's first publication in *The Journal of Bone and Joint Surgery* was on the use of maggots in the treatment of osteomyelitis his greatest aptitude was in the field of mechanics. He was one of the first orthopaedists to train himself in the use of power tools. He studied engineering principles, particularly in the field of stress analysis and applied these to bone surgery. He described several surgical procedures, including his operation for hallux valgus; his technique for the excision of the semilunar cartilages of the knee; his procedure for the treatment of interdigital neuromata; and prostheses for the hip, patella, tibial plateau, and, more recently, the acetabulum.

Duncan traveled far and wide, both as a student and a teacher in his own and allied professions. He made several trips to the blue-grass country of Lexington, Kentucky, where he helped pioneer the surgical treatment of bowed tendons in race horses which was so successful that it enabled animals, who might otherwise have been destroyed, to come back as winners.

Duncan had many friends south of the border. He learned to speak Spanish after he was fifty, and was host to many Latin American surgeons, many of whom spent several weeks as guests in his home while observing and assisting him at operations and on rounds. Termination of this little-known phase of his generosity deprives the United States of one of its most popular ambassadors.

In addition to his writings are his scientific movies produced at his own expense for the Audio-Visual Program. Principal subjects were reconstruction of badly damaged knee joints, mechanical stresses of lumbosacral stabilization, and interdigital neuroma, as well as a film concerning removal of a semilunar cartilage of the knee entitled "Take It Out Backwards."

We extend our sympathy to his wife, Dorothy Grant McKeever, whom he married in 1934, and to their three sons who plan to follow in their father's footsteps. The oldest, twins, twenty-four, Rex and Dick, are sophomores at Jefferson Medical School; and John, the youngest, twenty-one, is a pre-medical student at Wabash College. The McKeever family was united by a variety of mutual interests, among which were hunting turkey, deer, and duck; fishing; a goat ranch; and communication by ham radio when the children were away at school.

All who knew Duncan are appalled and saddened by what appears to be a senseless and untimely death. As one of his closest admirers summed it up "Duncan wasn't through working yet."

G.P.

Book Reviews

THE HAND: ITS ANATOMY AND DISEASES. By John J. Byrne, M.D., Springfield, Illinois, Charles C. Thomas, 1959. $10.50.

This book of 359 pages is essentially a review of a selected list of articles concerning the anatomy and the diseases of the hand. There are half a dozen references to the 1956 literature, but over 700 prior to that. No 1957 or 1958 articles are mentioned.

The book is divided into five sections: *Structure and Development, Infection, Trauma, Other Diseases, and Reconstructive Surgery.* Practically every lesion of the hand, whether congenital or acquired, is noted and a reference given. Under most headings there is very little discussion other than the factual reporting of the reference cited, and one is left to wonder what the author's own experience and feeling are in regard to some of the topics covered.

The section on anatomy is too superficial and elementary for a surgeon, and the drawings add nothing to the text. A reference to Kaplan's book or even to Ciba's give-away pamphlet with Netter's drawings will illustrate how clear and good anatomical drawings can be. Even these, however, cannot be compared with the classic pages of Sobotta or Spalteholz.

Infections of the hand are well covered, in fact they are overemphasized. Twenty-four pages and fifty references are devoted to just the bacteriology of hand infections, and similar space is then given to the surgery of infection.

Part III discusses the subject of trauma to various tissues, skin, tendons, nerves, bones, and joints. The author lists criteria for deciding when to do primary repair in fresh wounds. These criteria are apparently based on Flynn's work of some years past, although Flynn has altered these since 1956. Dr. Byrne's description of the technique of tendon repairs and some of the problems encountered in this part of surgery of the hand is succinct and well done. The part on tendon ruptures could stand more explanation. Under a subheading of *Baseball Finger* the author says "when there is a chip fracture of the distal interphalangeal joint, one should not immobilize in extension . . . the finger is maintained in a slight position of flexion." This is confusing. *Where is the fracture, and* what is a slight position?

The section on nerves is sketchy, and there is no reference to Seddon's classic studies of nerve suture and regeneration. The section on bones deals with fractures in an elementary way, and again, although thirty references are listed, there is no mention of Gedda's splendid study of Bennett's fracture. Kirschner gets three different spellings in just two pages.

There are 103 pages and 320 references in the next section called *Other Diseases.* The topics range from dermatological disturbances to Dupuytren's contracture, stenosing tenosynovitis, tumors, congenital deformities and vascular lesions, frostbite, and the painful states of causalgia and amputation neuroma. This is a difficult assortment to organize in any semblance of order. It certainly represents a lot of reading by the author. Burger's detailed German textbook, *Die Hand des Kranken,* is not mentioned in the bibliography.

Part V is entitled *Reconstructive Surgery.* Since only fifty pages are allotted to this, one would judge that the author feels that it is of relatively little importance. He gives 103 references to the literature and allots equal space—that is, seven pages—to the problems of skin reconstruction and to those of bones and joints. The entity of ischemic contracture gets the same amount of space; in this, there is no mention again of Seddon's splendid work on Volkmann's ischemic contracture.

The author has a pleasant style, the type face is easy to read and the publisher has printed it well on good paper, and the book is bound firmly. If the list of references were complete and brought up to date it would be a good index to the literature of the hand.

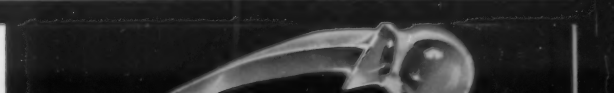
Joseph H. Boyes, M.D.

ORTHOPAEDICS: PRINCIPLES AND THEIR APPLICATION. Samuel L. Turek, M.D. Philadelphia, J. B. Lippincott Company, 1959. $22.50.

In recent years several excellent books on orthopaedics have been published, but no one can object to another volume which covers not only the strict field of orthopaedics but also some of the closely related material of other specialties.

The organization of the material is well done. The first part is concerned with the basic sciences as they apply to musculoskeletal tissues. Anyone who has been concerned with the teaching of this subject would wish that the student were thoroughly grounded in this basic material prior to presenting himself for the study of the many orthopaedic conditions. Part two deals with general orthopaedic conditions, metabolic diseases, congenital deformities, developmental diseases, neoplastic diseases, muscle diseases, bone and joint infections, as well as the orthopaedic aspects of neurology and vascular disease. Part three approaches orthopaedics from the regional standpoint. Part four concerns itself with radioactive isotopes and amputations, and is a fine addition to an orthopaedic text.

The book is written in clear readable style using the minimum number of words, illustrated



well by pictures and diagrams (many in color), liberally sub-titled and documented by references in close relation to the subject under discussion.

The surgical anatomy at the beginning of each section of the regional approach is helpful, not only for the student, but also for the surgeon who cannot refresh his memory too frequently in this important aspect of surgery. Much attention is also given to pathology in the disease section, with excellent photomicrographs, many in color. Examination and diagnostic methods are given much attention. Treatment is also well handled with only brief attention to the technique of operations.

This is distinctly a book for the student's and the practitioner's library. It should be at the practitioner's elbow to consult when troubled, and to read for pleasure and for learning, when leisure permits.

Denis S. O'Connor, M.D.

THE ESSENTIALS OF ROENTGEN INTERPRETATION. Lester W. Paul, M.D., and John H. Juhl, M.D. New York, Paul B. Hoeber, Incorporated, 1959. $25.00.

The many excellent textbooks of radiology now available range from a small volume for the medical student to the multiple-volume editions intended as reference sources for the specialist in radiology. It would therefore seem unlikely that a new textbook in this field could find an acceptable place in today's market. However, *The Essentials of Roentgen Interpretation* by Paul and Juhl more than accomplishes the authors' stated goal in writing the book: a presentation in a concise and readable form of the basic facts of roentgen interpretation that is of value to the specialty as a whole. The book is divided into six sections that comprise the major areas of roentgenographic interest, and in each section all of the common and most of the unusual conditions and diseases are discussed, with a concise listing of the positive roentgen findings and a discussion of differential diagnostic features. The technical information about the various types of roentgenographic examinations is limited to a brief description of what each entails for the patient.

The book contains 812 pages of text and 1,203 illustrations, which are prudently chosen with the aim of documenting the average, rather than the extreme, appearance of abnormality. The illustrations are of unusually good quality, and obviously were chosen with an eye toward good photographic reproduction, a feature that is not always common in textbooks but is the *sine qua non* of a roentgenography text.

From the roentgenologist's point of view, the section on the abdomen and gastro-intestinal tract is valuable and seems to be a rather neat compilation of present-day rationale of roentgen diagnosis. This section covers a number of current subjects that the authors realize are controversial and, as in the other sections, the bibliography contains pertinent references to the recent literature.

Of particular interest for readers of this Journal is the excellent section on the osseous system, which comprises approximately a quarter of the book. The subsection on anomalies and dysplasias is terse but inclusive, with excellent illustrations of both the common and the bizarre forms of these abnormalities. The subsections on bone tumors and demineralization of bone are equally well presented.

This book should achieve a place of merit and be of great usefulness to roentgenologists, to physicians who are beginning the study of a medical or surgical specialty, and to those who wish a brief but thorough reference to the roentgen diagnostic features in their specialty or sphere of interest.

J. R. Dreyfuss, M.D.

Richards

FEMORAL PROSTHESES

(SMo STAINLESS)

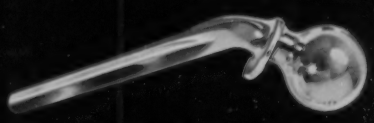
for every requirement

Richards Femoral Prostheses are solid forged, precision machined of SMo stainless, electropolished for optimum tissue tolerance. Offer maximum ductility, maximum strength, mirror finish and hollow concentric head for reduced weight. These popular forms available with specially designed rasps and broach to facilitate insertion. Teflon faced drivehead assures against damage to polished, precision head, producing smooth motion against the acetabulum, precludes possible contamination to surface of prosthesis head. Packaged in Kane Sterifabric Sterilizing Case.

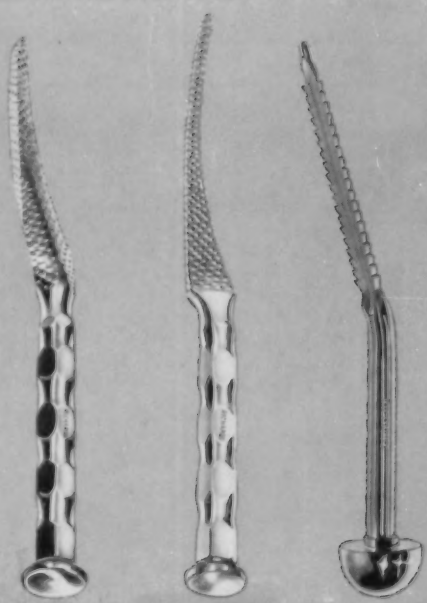
Write For Full Information

F. R. Thompson Type Hip Prosthesis — Popular choice of surgeons because of its ease of insertion and good results.

Moore Type Hip Prosthesis — Holes in stem allow bone growth through the prosthesis (sometimes filled with bone crumbs before insertion).



Eicher Type Hip Prosthesis — Tri-finned shaft, available in two diameters. Shaft length 5½".



Rasp for F. R. Thompson type prosthesis.

Rasp for Moore type prosthesis.

Broach for Eicher type prosthesis.

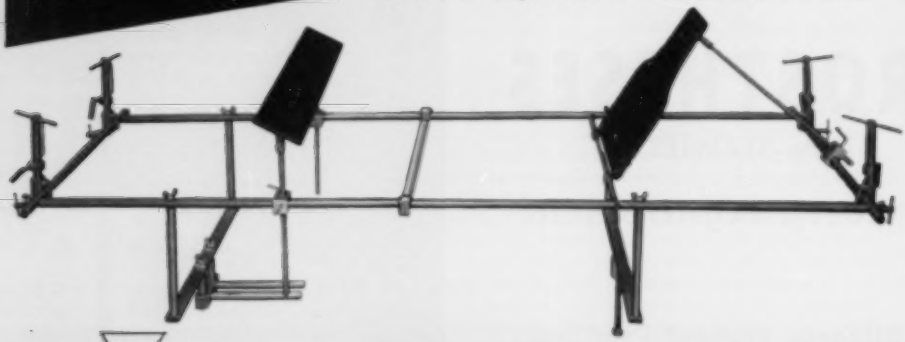
Teflon Faced Drivehead — Anodized aluminum body. Totally inert driving face of Teflon. Concave face fits all heads. May be autoclaved.

Richards MANUFACTURING COMPANY

756 Madison Avenue, Memphis 3, Tenn.

Complement to the Surgeon's Skill

RS Attachment! Fits any Chick Table. Designed with the patient and the doctor in mind! Gives patients comfort. Provides doctors with complete freedom in which to work. Ideal for application of casts in cervical spine, scoliosis, and other hyper-extension cases. RS Attachment fits DVHI, Dr. Virgin Tables, and Bell Tables, remarkable orthopedic and surgical tables available only under the CHICK trademark. Write today for additional information.



GILBERT HYDE **CHICK** COMPANY

Main Office and Plant: 821-75th Avenue, Oakland 21, Calif.
General Sales Office: 5 Broadway, East Paterson, New Jersey
In Canada: J. F. Hartz Co., Ltd., 34 Grenville St., Toronto

Manufacturers and Distributors of Hospital Orthopaedic and Fracture Equipment

PRESTON *A Complete Line* **FOR PHYSICAL THERAPY AND REHABILITATION**

REHABILITATION: **Bicycle Exercisers; Restorator; Walking Parallel Bars and Exercise Staircases** of various designs; Posture Mirrors; Gymnasium Mats; **Shoulder Wheels** with or without height adjustment; **Elgin Progressive Resistance Exercise Units;** Quadriceps Boots; Dumbbells; Chest Pulley Weights; **Standing (Tilt) Tables; Stryker Turning Frames.**

HAND THERAPY DEVICES: Kanavel Table; Manuflex; Wrist Exercisers; Complete Line of **Bunnell Splints;** Theraplast.

TRACTION: Many types of Cervical and Pelvic Traction Sets; **Hausted Tractionaid.**

HYDROTHERAPY — ELECTROTHERAPY: **Whirlpools;** Paraffin Baths; **Hydrocollator;** Ries Moistaire; **Medcolator** and other Low-Volt Generators; Infrared Lamps; Bakers; **Birtcher Ultrasonic Generators.**

Everest & Jennings Wheelchairs; Commodes; **Walkers** and **Walking Aids for every need; Hydraulic Patient Lifters; Largest selection of Treatment Tables.**

Complete Line of Cerebral Palsy Equipment — Speech Therapy — **Self Help Devices for the Handicapped.**

CONVENTION NEWS

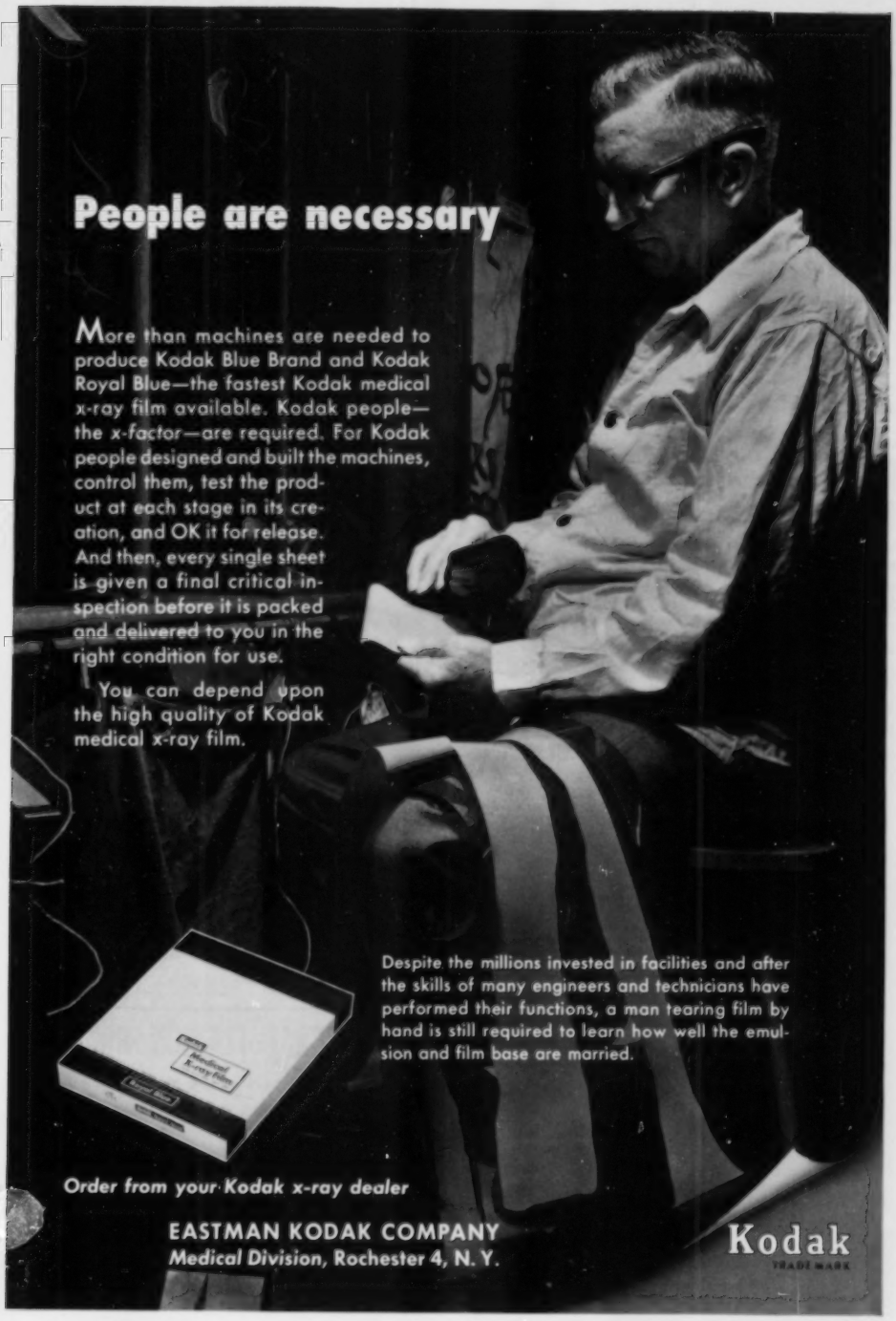
At the Chicago Convention be sure to see

- ALL Bunnell Hand and Finger Splints including three all-new extension splints
- New Motorized Bicycle—only $79.95
- The fool-proof Jamar Hand Dynamometer —for positive grip strength evaluation
- And Selected Exercise Equipment, Whirlpools and Traction Units

at Preston Booth #10 and #11

We supply *ALL EQUIPMENT* for setting up a complete Rehabilitation Program. Suggested layouts and equipment lists are available to you through *OUR FREE PLANNING SERVICE.*

J. A. PRESTON CORP., 175 FIFTH AVENUE, NEW YORK 10, N. Y.


People are necessary
More than machines are needed to produce Kodak Blue Brand and Kodak Royal Blue—the fastest Kodak medical x-ray film available. Kodak people—the x-factor—are required. For Kodak people designed and built the machines, control them, test the product at each stage in its creation, and OK it for release. And then, every single sheet is given a final critical inspection before it is packed and delivered to you in the right condition for use.
You can depend upon the high quality of Kodak medical x-ray film.
Despite the millions invested in facilities and after the skills of many engineers and technicians have performed their functions, a man tearing film by hand is still required to learn how well the emulsion and film base are married.
Order from your Kodak x-ray dealer
EASTMAN KODAK COMPANY
Medical Division, Rochester 4, N. Y.
Kodak
TRADE MARK

To serve your complete needs in
ORTHOPEDIC, PROSTHETIC
and SURGICAL APPLIANCES

house of BIDWELL inc.

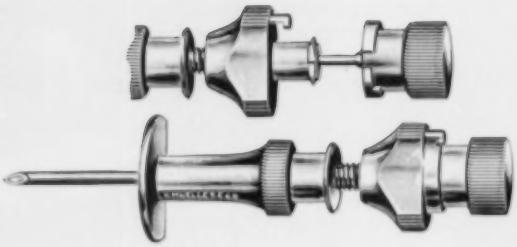
535 N. 27th Street
Milwaukee 8, Wis.

1134 Regent St.
Madison, Wisconsin

University of Illinois Sternal Puncture Needle . . .

SIMPLIFIES MARROW ASPIRATION

Specially constructed Luer lock hub and a knurled edge on a large stylet head — plus a stylet which locks in place — have made the University of Illinois Needle the instrument of choice for marrow aspirations from the sternum. These features also make it easy to utilize such alternative sites of bone marrow puncture as the iliac crest, vertebra, tibia, femur, and internal malleoli. Adjustable for depth, the needle may also be used for infusions into the marrow, if desired. Usable with standard glass tip or Luer lock type syringes.



SU-21000 • University of Illinois Sternal Needle. Stainless. 15g x 1", each. **$14.50**

For complete information ask your V. Mueller representative, or write . . .

V MUELLER & CO.

Fine Surgical Instruments and Hospital Equipment

330 South Honore Street • Chicago 12, Illinois

DALLAS • HOUSTON • LOS ANGELES • ROCHESTER, MINN.

Armamentarium

WILLIAMS LUMBO-SACRAL FLEXION BRACE

Orders filled at the request of members of the profession only

Measurements: 1. Chest (about 4" below nipple line); 2. Waist (at navel line); 3. Pelvic (½ distance between greater trochanter and crest of ilium); 4. Seventh cervical spinous process to the prominence of Coccyx.

ALL ORTHOPAEDIC APPLIANCES

MILLER BRACE CO.

3902 Gaston Avenue Dallas, Texas

For prompt service use our P. O. Box 7902

Custom Built: Braces—Surgical Supports—Splints—Arch Supports—Folding Wheel Chairs—Aluminum Adjustable Crutches—Airfoam Head Halter Sets—Airfoam Rib Binders—Canes, Etc.

HYDROCOLLATOR

Steam PACK

MOIST HEAT

Now moist heat can be applied conveniently, effectively and with a minimum investment in equipment. No dripping, no wringing, no repeated applications. Each application gives at least 30 minutes effective moist heat. The Steam Pack is merely heated in water, wrapped in a towel, and applied. Standard equipment in leading hospitals and clinics across the nation.

DESCRIPTIVE MATERIAL ON REQUEST

CHATTANOOGA PHARMACAL CO., INC.

CHATTANOOGA 5, TENNESSEE

MASTER UNITS

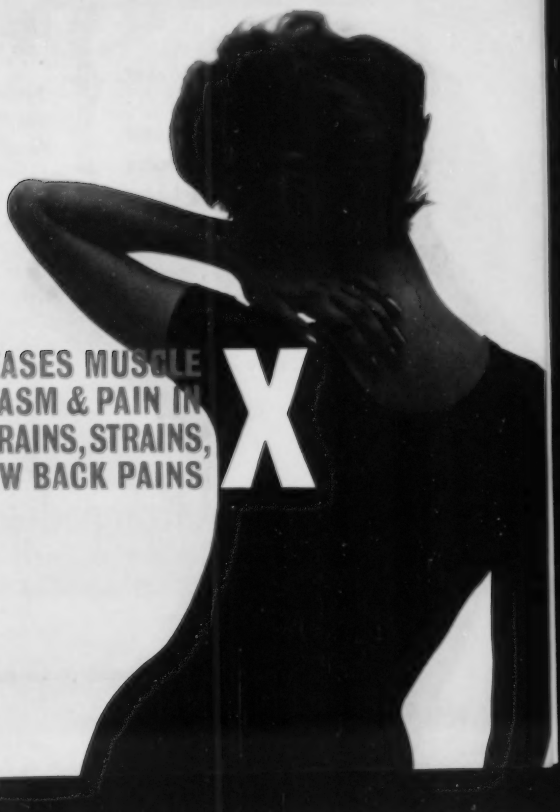
Four all stainless steel models to meet the various requirements in hospitals, clinics, physicians' offices, and patients' homes. Automatically maintains Steam Packs in water at proper temperature — constantly ready for immediate use. No plumbing used.

D-3 2 Pack

E-1 4 Pack

SS — 6 Pack

M-2
12 Pack Mobile Unit



ANNOUNCING SCHERING'S NEW MYOGESIC[X]

RELA™ CARISOPRODOL

–EASES MUSCLE SPASM & PAIN IN SPRAINS, STRAINS, LOW BACK PAINS

X

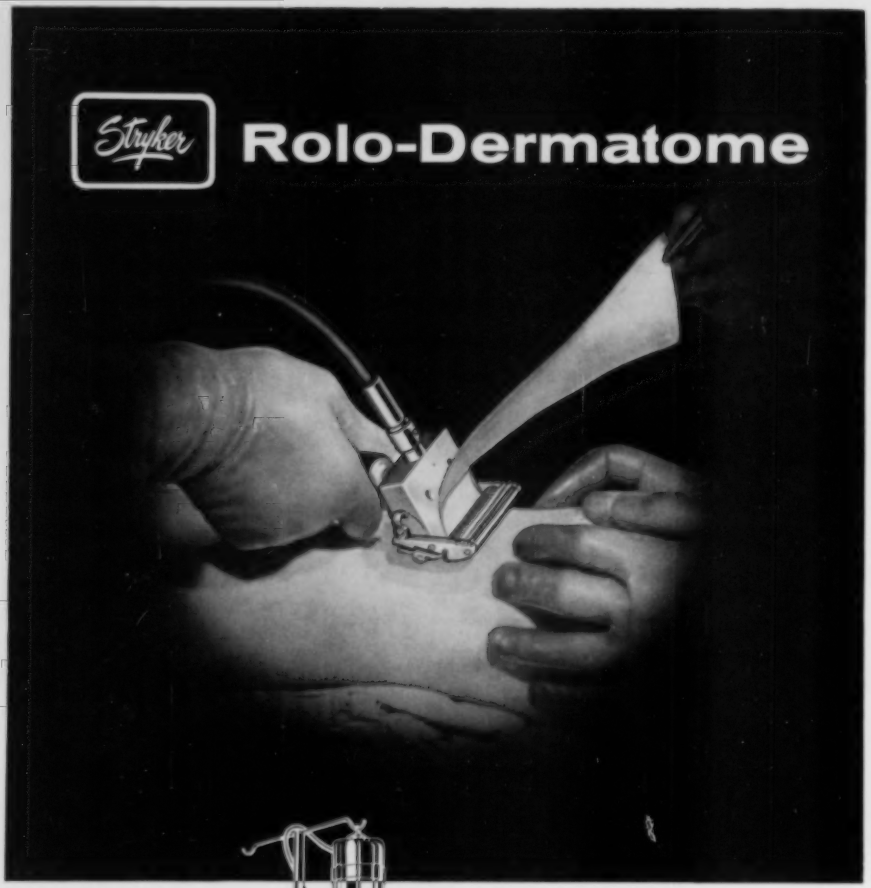
[X]**MYOGESIC**
muscle relaxant – analgesic

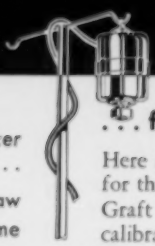
Schering

H-378

Stryker

Rolo-Dermatome





. . . for Precision Skin Grafting

Here is Orthopedic Frame's answer to the demand for the finest grafting instrument possible.
Graft thickness is accurately obtained by a single calibration lever on the right side of the instrument. Minimum setting, 5/1000"; maximum setting, 30/1000". Adjustable graft widths, 3", 2¼", 1½ inches.
Two rollers in front and one behind the oscillating blade eliminate tension as the skin comes in contact with the disposable blade. Few moving parts . . . two year guarantee.

Handpieces for the Stryker Electro-Surgical Unit . . .

- Bone Saw
- Rolo-Dermatome
- Sagittal Saw
- Pencil-Grip Burring Handpiece
- Bone Drill Handpiece
  Automatic Screwdriver
  Colorguide Drill
  Jacobs Chuck
- Colorguide Screw Case

To give body and original shape to skin grafts, use Stryker Skin Tape.

Stryker Manufacturing Quality Medical Equipment for 15 years

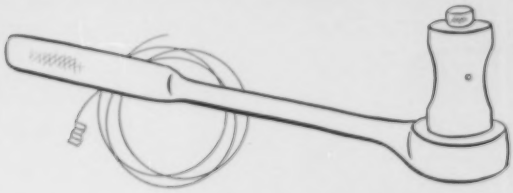
Orthopedic Frame Company

420 ALCOTT STREET • KALAMAZOO, MICHIGAN

Exclusive Agent for Export: Schueler & Co., 75 Cliff St., N. Y.

KASTOFF® — *The Modern Method of Cast Removal*

See our exhibit at American Academy of Orthopedic Surgeons Meeting—Chicago, January 23–28, Booth #49 (with V. Mueller & Co.)



SIMPLE
SAFE
SURE

Instrument..**$17.50**
Removel Wires.....
$3.60 per doz.
(In 6 doz. lots).....
$2.95 per doz.

KASTOFF, INC. **4924 Greenville Avenue, Dallas 6, Texas**

The Ruth Jackson Cervical Collar

of "CERVICAL SYNDROME"
BY
RUTH JACKSON, M.D., F.A.C.S.

An all-purpose plastic collar for extension, stabilization, and the recommended flexed, or straight neck and "chin tucked in", position indicated in whiplash sprains and other cervical conditions. Completely adjustable for proper fitting, position and support with minimal pressure at the ridge of the trapezius. Available to brace makers.

Literature and reprint on request

FLORIDA MANUFACTURING CORPORATION
TERRY MOORE, PRESIDENT
BOX 449 • DAYTONA BEACH, FLORIDA

*For Congenital Dislocation of the Hip—***ORDER**

FREJKA
ABDUCTION PILLOW SPLINTS

with the new Wet-Proof pillow

3 SIZES:
Small—6" pillow splint (up to 4 months)
Medium—9" pillow splint (4 to 8 months)
Large—12" pillow splint (over 8 months)
Available in Pink, Yellow & White

PHONE—Midway 4-6874

MINNESOTA ORTHOPEDIC SERVICES
1963 Carl Street **St. Paul 13, Minn.**

PRICE

With
Extra
Halter
$12.50

POSTPAID

NO MORE

because

VITAGENIC

THE PRESCRIPTION-BUILT
EXTRA-FIRM MATTRESS
NEEDS NO
INTERPOSED BOARD

Physicians and orthopaedic specialists know that a bedboard—even one that is interposed in a mattress—is only an attempt to correct a structural weakness and inadequacy in the mattress itself. The VITAGENIC by AIRELOOM requires no "make-shift" additions.

THE VITAGENIC is a mattress and box spring combination made according to the exact specifications of registered orthopaedic specialists. It is an important—almost essential—adjunct to your plan of treatment for pain of backache or sacroiliac injury. You prescribe the exact degree of firmness necessary for your patient as well as any other features required for treatment or comfort.

CURLED HAIR FOR RESILIENT FIRMNESS

Firm body support without undue pressure on nerve points or interference with circulation.

HAND-STITCHED SAG-FREE SIDEWALLS

Sag-free edges allow patients with back injuries to get in and out of bed with less effort and greater stability. 12% more usable sleeping area.

THE VITAGENIC IS GUARANTEED FOR 15 YEARS

For full details on the prescription-built VITAGENIC, contact:

Aireloom

BEDDING COMPANY
1119 Westminster Ave., Alhambra, Calif.
created by King Karpen—established 1951

CORNELL UNIVERSITY MEDICAL COLLEGE

ANNOUNCES THE FIFTH ANNUAL POSTGRADUATE COURSE IN

The Treatment of Fractures and Other Trauma

at

The Hospital for Special Surgery and New York Hospital — Cornell

JUNE 13-18, 1960

This six-day course is given annually by members of the Cornell University Medical College faculty, serving on the staff at the Center hospitals. In addition to fractures and dislocations, the program offers a comprehensive review of the treatment of other traumatic conditions, including burns, shock, hand injuries, and trauma to abdomen, chest and nervous system.

Living accommodations will be available to postgraduate students and their wives in the Cornell Medical Student Residence, Olin Hall, at $3.00 per person per night.

TUITION: $150.00 — Enrollment limited
For further information write to:

DR. PRESTON A. WADE
Cornell University Medical College
1300 York Avenue, New York 21, New York

THE MONTREAL GENERAL HOSPITAL

Orthopaedic Course

Examination preparation for Orthopaedic Certification or Fellowship in the Royal College of Surgeons of Canada given by members of the Department of Orthopaedic Surgery of The Montreal General Hospital.

The first part of the course commencing in March consists of a weekly reading list covering consecutive phases of orthopaedic surgery, and a written question which is corrected and returned to the candidate with comments. General surgery can be included for the Fellowship candidates if desired. The second part of the Course is optional and consists of practical teaching in Basic Sciences, General Surgery and Orthopaedic tutorials, starting in Montreal in August.

Tuition Fees:

Part I—$150.00 Part II—$75.00

COMBINED COURSE—$225.00

Apply to: The Registrar, Postgraduate Board
THE MONTREAL GENERAL HOSPITAL
1650 Cedar Avenue, Montreal 25, P. Q., Canada

ACTIVE HAND and FINGER SPLINTS

Instruments for Hand Surgery and Treatment

AS DESIGNED BY

Dr. STERLING BUNNELL

Carried in stock for immediate delivery

- Knuckle bender splint
- Radial palsy splint
- Finger extension splint, etc.
- Hand Drill w/Jacobs chuck
- Tendon stripper set
- Gig pull-out suture and

other special instruments for bone and joint surgery

▶ Write for catalogue — postage prepaid ◀

H. WENIGER

Established 1907

New Address

70-12th Street, San Francisco 3, Cal.

HAS YOUR

ADDRESS

CHANGED?

PLEASE NOTIFY US PROMPTLY

IS YOUR

ADDRESS

GOING TO CHANGE?

PLEASE NOTIFY US IN ADVANCE

• • •

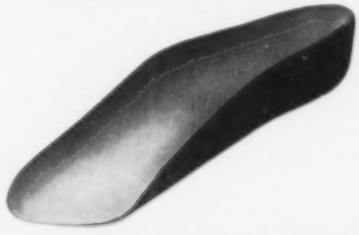
WHEN YOU SEND US A CHANGE OF ADDRESS PLEASE GIVE US

both old and new addresses

New Comfort For Arthritic Patients!

KLEISTONE'S

Lynco #57 CUSHION MOLD



Made of resilient cellular rubber, covered on top with soft, pliable leather. The bottom is not covered, so that this cushion may be altered easily, by additions or reductions to fit the individual case.

Write for full details

KLEISTONE RUBBER COMPANY, Inc.

Established 1919

Warren, Rhode Island, U. S. A.

1959 INSTRUCTIONAL COURSE LECTURES

of The American Academy of Orthopaedic Surgeons

HAVE you felt the need for refreshing your knowledge and learning new techniques but believe you have no time in your busy practice to attend postgraduate courses? You can keep up-to-date by studying the new 1959 INSTRUCTIONAL COURSE LECTURES selected by the Academy from those given at the 1959 meeting as having greatest current interest. Here is just a sampling of the authoritative, clinical information which you can extract from some of the 335 pages in this atlas-size, $8\frac{1}{2}$" x $10\frac{3}{4}$" book:

- **Diagnosis and Treatment of Ankle Injuries Sustained in Sports** *(T. B. Quigley, M.D.)*
- **Surgery of the Paralytic Hand** *(Daniel C. Riordan, M.D.)*
- **Treatment of Clubfoot** *(John McCauley, Jr., M.D.)*
- **Diagnosis, Treatment, and Rehabilitation of the Industrial Low Back Cripple** *(Chester C. Schneider, M.D.)*
- **The Moore Self-Locking Vitallium Prosthesis in Fresh Femoral Neck Fractures** *(Austin T. Moore, M.D.)*

The C. V. Mosby Company

3207 Washington Boulevard, St. Louis 3, Mo.

Please send me on 10 day approval a copy of the 1959 INSTRUCTIONAL COURSE LECTURES, priced at $16.00.

☐ **Payment enclosed** ☐ **Charge my account**

.....M.D.

Address.....

City.....*Zone*....*State*.....JBS-1-60

List of Advertisers

JANUARY 1960

	PAGE
Aireloom Bedding Company	60
C. H. Alden Shoe Company	26
American Sterilizer Company	27
Austenal, Inc.	17, 18, 19, 20
The Berivon Company	50, 51
Bowen & Company, Inc.	6
S. H. Camp and Company	24
Chattanooga Pharmacal Co., Inc.	57
G. W. Chesbrough Co.	44
Gilbert Hyde Chick Company	54
Codman & Shurtleff, Inc.	40
Cornell University Medical College	60
DePuy Manufacturing Company, Inc.	31
Eastman Kodak Company	55
J. Edwards Shoe Company	42
Fillauer Surgical Supplies, Inc.	15
Florida Manufacturing Corporation	59
Freeman Manufacturing Company	33
Hack Shoe Company	46
J. E. Hanger, Inc.	14
Herbst Shoe Manufacturing Company	38
House of Bidwell, Inc.	56
Kastoff Company	59
Kleistone Rubber Company	61
Lederle Laboratories, Division of American Cyanamid Company	8, 9
McNeil Laboratories, Inc.	16, 49
M. J. Markell Shoe Company Inc.	30
Medical Research Specialties	48
Miller Brace Co.	56
Minnesota Orthopedic Services	59
The Montreal General Hospital	60
The C. V. Mosby Company	61
V. Mueller & Co.	56
Orthopedic Equipment Co.	4
Orthopedic Frame Company	34, 58
J. A. Preston Corp.	54
Richards Manufacturing Company	47, 53
I. Sabel, Inc.	10
Schering Corporation	28, 29, 57
Chas. A. Schmidt Surgical Instrument Company	32
Spencer, Incorporated	41
E. R. Squibb & Sons	11, 12, 13
Charles C. Thomas	52
Truform Anatomical Supports	7
Wallace Laboratories	36, 37, 43
H. Weniger	61
Winthrop Laboratories	21
Wright Manufacturing Company	45
Wyeth Laboratories	5, 25, 35, 39
Zimmer Manufacturing Company	22, 23

In answering advertisements, please mention *The Journal of Bone and Joint Surgery.*

ADDRESSES OF CONTRIBUTORS

JANUARY 1960

HENRY H. BANKS, M.D., 721 Huntington Avenue, Boston 15, Massachusetts
WILLIAM H. BLODGETT, M.D., 5447 Woodward Avenue, Detroit 2, Michigan
GEORGE E. BROMAN, M.D., St. Joseph's Hospital, Denver, Colorado
CHARLES G. CHILD, III, M.D., University Hospital, Ann Arbor, Michigan
O. DONALD CHRISMAN, M.D., 264 Elm Street, Northampton, Massachusetts
ROSE ROBYNS COELHO, Ph.D., Children's Hospital, Boston 15, Massachusetts
COMMANDER ADOLPHUS W. DUNN, U.S. Naval Hospital, Great Lakes, Illinois
A. W. FARMER, M.D., Suite 325, Medical Arts Building, Toronto 5, Ontario, Canada
PAUL FIELD, M.B., B.S., Hospital for Sick Children, Toronto, Ontario, Canada
J. EDWARD FLYNN, M.D., 412 Beacon Street, Boston, Massachusetts
ROBERT FREIBERGER, M.D., 535 East 70th Street, New York 21, N. Y.
HAROLD M. FROST, M.D., Henry Ford Hospital, Detroit 2, Michigan
JAMES H. GRAHAM, M.D., 120 Melrose Street, Melrose 76, Massachusetts
WILLIAM T. GREEN, M.D., 300 Longwood Avenue, Boston 15, Massachusetts
S. J. HOUTZ, M.S., 5447 Woodward Avenue, Detroit 2, Michigan
REX J. HOWARD, M.D., 930 Fifth Avenue, Fort Worth 4, Texas
L. P. KLEUSER, M.D., 930 Fifth Avenue, Fort Worth 4, Texas
LOREN J. LARSEN, M.D., Franklin Hospital, San Francisco 8, California
C. A. LAURIN, M.B., B.S., c/o A. W. Farmer, M.D., Suite 325, Medical Arts Building, Toronto 5, Ontario, Canada
ALBERT F. MARTIN, M.D., 3123 Majestic Drive, Salt Lake City, Utah
JAMES A. NICHOLAS, M.D., 150 East 77th Street, New York 21, N. Y.
BENJAMIN E. OBLETZ, M.D., 685 Delaware Avenue, Buffalo 9, New York
ROY P. PETERSON, Ph.D., The School of Anatomy, Cambridge University, England
ROBERT B. SALTER, M.D., M.S., F.R.C.S.(C), Medical Arts Building, Toronto, Ontario, Canada
EDWIN R. SCHOTTSTAEDT, M.D., Franklin Hospital, San Francisco 8, California
GERHART S. SCHWARZ, M.D., Presbyterian Hospital, 622 W. 168th Street, New York 32, N. Y.
MILDRED TROTTER, Ph.D., Washington University, School of Medicine, St. Louis 10, Missouri
JOHN T. WILSON, M.D., Boston City Hospital, Boston, Massachusetts
PHILIP D. WILSON, M.D., 535 East 70th Street, New York 21, N. Y.

RUMFORD PRESS
CONCORD, N. H.